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INTRAOCULAR TENSION IN CASES OF SARCOMA OF THE CHOROID AND CILIARY BODY

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This study of the intraocular tension in eyes containing new growths was undertaken in an endeavor to determine the diagnostic value of this test in the differential diagnosis between a serous detachment of the retina and one due to an intraocular neoplasm.

The occurrence of secondary glaucoma as a late manifestation of intraocular sarcoma is universally recognized, but the behavior of the intraocular tension in the early stages of the disease is not well known. The literature on this phase of the subject is confusing, for some authors have made no mention of it, while one stated: "Tension is a valuable diagnostic point; in retinal detachment due to a serous collection it is generally decreased; in choroidal sarcoma it is usually normal at first and becomes increased later." [1] Still another [2] stated: "Too much reliance cannot be placed upon the tension of the eyeball as a distinguishing sign between sarcoma and retinal detachment because intraocular tension may be unaltered in each instance, although as C. Devereux Marshall has shown it is probably never diminished in undoubted cases of choroidal sarcoma while it may be reduced in cases of sarcoma of the ciliary body."

The earliest reference to hypotony in cases of intraocular neoplasm was made by Laurence [3] in 1866. He reported a case in which the eyeball was very soft. The condition was not diagnosed until the eye was enucleated after the patient had previously been subjected to iridectomy for glaucoma with subsequent hypotony. Laurence cited this case

From the Institute of Ophthalmology, Presbyterian Hospital.

Read before the New York Academy of Medicine, Section of Ophthalmology, May 16, 1938.

1. Ball, J. M., cited by Findlay, E. K.: Tumors of the Choroid, in Wood, C. A.: The American Encyclopedia and Dictionary of Ophthalmology, Chicago, Cleveland Press, 1914, vol. 3, p. 2180.

2. de Schweinitz, G. E.: Diseases of the Eye, ed. 10, Philadelphia, W. B. Saunders Company, 1924, p. 405.

3. Laurence, J. Z.: Ophth. Rev. **2**:378, 1866.

as an exception to von Graefe's dictum that "the degree of tension of the eyeball is a guide to diagnosis." In commenting on this unusual finding, he stated: "Mr. Nunneley of Leeds told me he had frequently met with soft globes in the first stage of malignant disease of the eyeball." The first real study on this subject was made in 1889 by Marshall,[4] who concluded that the intraocular tension was seldom lowered in globes containing new growths. He found the tension increased in 66.6 per cent, normal in 30.3 per cent and lowered in 3.03 per cent. Carrying his studies still further, he[5] reported in 1896 on 100 cases in which he "compared the pathological state with the condition they presented before excision." From these studies he concluded that "diminished tension by no means uncommonly occurs when the ciliary body is involved, but I have been unable to find an undoubted case in which the tension has been reduced when the choroid only is affected."

In 1898 Leber and Krahnstöver[6] reviewed the literature on hypotony in cases of intraocular sarcoma. They reported on 37 such cases, adding the reports of several of their own to those previously recorded. No attempt was made to measure the intraocular tension of both eyes, and the series of cases was largely made up of those in which phthisis bulbi was a late manifestation. In 11, there was profound hypotony without actual shrinkage of the eyeball, but the authors contended that these eyes were in a transitional stage. They showed that phthisis bulbi is usually not the result of a perforation and concluded that the hypotony and shrinkage of the eyeball come from a chronic plastic inflammation due to bacteria rather than from the toxins formed by necrosis of the tumor. They speculated that the frequency of endogenous infection may be due to the fact that necrotic tumor tissue forms a good medium for bacterial growth.

Franz[7] in 1920 was the first to record the intraocular tension of both eyes in his report on hypotony as an early manifestation of choroidal sarcoma. He reported the detailed observations in 3 such cases and concluded from his study that the lowered intraocular tension was not due to bacterial infection, uveal inflammation or necrosis. He discussed the causes of hypotony in cases of detachment of the retina and stated that the same causative factors were responsible for its occurrence in association with tumor. The tension of the affected eye was 6 mm. of mercury lower than that of the other eye in 1 of his cases, 8 mm. in 1 case and 12 mm. in another.

In the same year Francis[8] reported an interesting case of intraocular sarcoma in which the patient was followed for five years on account

4. Marshall, C. D.: Roy. London Ophth. Hosp. Rep. **15**:57, 1889.
5. Marshall, C. D.: Tr. Ophth. Soc. U. Kingdom **16**:155, 1896.
6. Leber, T., and Krahnstöver, A.: Arch. f. Ophth. **45**:164, 1898.
7. Franz, G.: Klin. Monatsbl. f. Augenh. **64**:348, 1920.
8. Francis, L. M.: Am. J. Ophth. **3**:872, 1920.

of a macular lesion. When he was first seen, the intraocular tension was 20 mm. of mercury, while at the time of enucleation it was only 5 mm. Pathologic examination failed to reveal a perforation, and the tumor was located in the choroid near the optic disk. So while the existence of hypotony in association with intraocular sarcoma has been known for many years, no recent attempt has been made to establish the frequency with which it occurs in the first stage of the disease.

ANALYSIS OF CASES

No case was considered in this analysis unless the clinical diagnosis was confirmed by a pathologic examination of the enucleated eye. Tonometric readings with a Schiötz tonometer were made on both eyes in every instance. Fifty-five cases of sarcoma of the choroid and the ciliary body have been analyzed, and the results are shown in table 1.

In 9 of the 15 cases in which the intraocular tension of the affected eye was higher than that of the sound eye, the disease had progressed

TABLE 1.—*Comparative Tension of the Affected Eyes*

Tension	No. of Cases	Percentage
Higher	15	27.3
Equal	11	20.0
Lower	29	52.7

to the second stage, as there was definite secondary glaucoma present. With the exclusion of these 9 cases from consideration, it was found that the intraocular tension was either equal to or less than that of the fellow eye in 86.9 per cent of the remaining 46 cases. A definite lowering of the intraocular tension was recorded in 63 per cent of these cases. The difference in the tonometric readings for the two eyes varied from 2 to 10 mm. of mercury, the average being 5 mm. The average tension in these 46 cases was 11.9 mm. of mercury. It is interesting to note that in a previous report Macnie and I [9] found the average intraocular tension in 55 cases of serous detachment of the retina to be 11.1 mm. of mercury. In none of these cases in which a lowered intraocular tension was found had the condition progressed through the glaucomatous state into one of phthisis bulbi. In every instance the diagnosis was made prior to the onset of glaucoma, i. e., in the first stage of the disease. It can therefore be said that in at least from 60 to 65 per cent of cases of sarcoma of the choroid and ciliary body a lowered intraocular tension is manifested in the early stages of the disease.

The 55 cases studied here were grouped according to the location of the primary site of the tumor, as shown in table 2.

9. Dunnington, J. H., and Macnie, J. P.: Detachment of the Retina: Operative Results in One Hundred and Fifty Cases, Arch. Ophth. **13**:191 (Feb.) 1935.

TABLE 2.—*Classification of Cases on Basis of Primary Site of Tumor*

Primary Site	No. of Cases	Tension		
		Higher	Equal	Lower
Choroid	47	14 (29.8%)	8 (17%)	23 (53.2%)
Ciliary body	8	1 (12.5%)	2 (37.5%)	4 (50%)

TABLE 3.—*Analysis of Fifty-Five Cases of Sarcoma of the Choroid and the Ciliary Body*

Case No.	Eye Involved	Tension		Location of Tumor
		O. D.	O. S.	
Group 1: Higher Intramuscular Tension in the Affected Eye				
1	O. S.	12	13	Choroid
2	O. D.	27	23	Choroid
3	O. S.	15	18	Choroid
4	O. D.	18	16	Choroid
5	O. S.	12	14	Choroid
6	O. D.	23	19	Choroid
7	O. S.	19	70	Choroid
8	O. S.	15	28	Choroid
9	O. S.	12	33	Choroid
10	O. S.	10	52	Choroid
11	O. D.	44	18	Choroid
12	O. D.	70	16	Choroid
13	O. S.	16	49	Choroid
14	O. S.	15	48	Choroid
15	O. S.	12	33	Choroid
Group 2: Equal Intraocular Tension in Both Eyes				
16	O. S.	10	10	Choroid
17	O. D.	10	10	Choroid
18	O. D.	8	8	Choroid
19	O. D.	14	14	Choroid
20	O. D.	14	14	Choroid
21	O. D.	20	20	Choroid
22	O. S.	8	8	Choroid
23	O. S.	20	20	Choroid
24	O. D.	15	15	Ciliary body
25	O. D.	10	10	Ciliary body
26	O. D.	8	8	Ciliary body
Group 3: Lower Intraocular Tension in the Affected Eye				
27	O. D.	8	14	Choroid
28	O. D.	8	10	Choroid
29	O. S.	18	15	Choroid
30	O. S.	20	12	Choroid
31	O. S.	16	12	Choroid
32	O. D.	7	13.5	Choroid
33	O. D.	10	12	Choroid
34	O. S.	16	12	Choroid
35	O. S.	14	9	Choroid
36	O. D.	10	16	Choroid
37	O. D.	5	14	Choroid
38	O. D.	12	14	Choroid
39	O. S.	17	10	Choroid
40	O. D.	11	14	Choroid
41	O. D.	7	13	Choroid
42	O. D.	14	18	Choroid
43	O. S.	12	8	Choroid
44	O. D.	12	14	Choroid
45	O. D.	8	13	Choroid
46	O. S.	23	12	Choroid
47	O. S.	16	7	Choroid
48	O. D.	18	23	Choroid
49	O. S.	18	14	Choroid
50	O. D.	18	23	Choroid
51	O. S.	18	14	Choroid
52	D. S.	12	8	Ciliary body
53	O. D.	9	12	Ciliary body
54	O. D.	8	14	Ciliary body
55	O. S.	20	10	Ciliary body

A study of the figures in table 2 reveals that of the 47 cases in which the disease was diagnosed as being of choroidal origin there was a lower intraocular tension in the affected eye in 25 (53.2 per cent). While in some of the cases secondary involvement of the ciliary body may have been present, in many of them the lesion was confined entirely to the choroid.

The number of cases of primary involvement of the ciliary body is too small to justify any comment, unless the relative infrequency of increased intraocular tension in this group is worthy of note, having occurred in only 1 of the 8 cases.

SUMMARY

Fifty-five cases of intraocular sarcoma have been analyzed in an endeavor to determine the frequency as well as the manner in which the intraocular tension is affected by the disease. In 9 of these cases definite secondary glaucoma had intervened.

In 29 (63 per cent) of the remaining 46 cases, the tension of the affected eye was found to be lower than that of the sound eye. In 11 (23.9 per cent) the intraocular pressure of the two eyes was equal, while in 6 (13.1 per cent), the affected eye showed a slightly higher tension. The amount of lowering of the intraocular tension varied from 2 to 10 mm. of mercury, the average being 5 mm. This decrease in pressure was encountered just as frequently when the disease originated in the choroid as when it started in the ciliary body. It is therefore felt that an initial drop in the intraocular tension is the rule rather than the exception in the early stages of sarcoma of the choroid and ciliary body.

Dr. John M. Wheeler allowed me to incorporate the data for many of his cases in this report.

CLINICAL SIGNIFICANCE OF THE RETINAL CHANGES IN LEUKEMIA

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Since Liebreich's description of the retinal changes in leukemia, numerous ophthalmologists have recorded their experiences in contributions which take into consideration the description, the incidence, the prognostic significance and the histologic picture of the various ocular lesions observed in the several types of this condition.

This report includes the results of a study of the retinal changes and the laboratory findings in 22 cases in which a diagnosis of leukemia was made by the members of the medical department of the Temple University Medical School. The clinical diagnosis was confirmed in 9 cases at autopsy and in 7 additional cases by biopsy. None of the eyes was studied microscopically. The ophthalmologic and clinical data in these cases conform in most instances to the observations of Wagener,[1] Goldback [2] and Frank.[3] The objects of this report are to discuss the ophthalmologic diagnosis and prognosis of leukemia and to call attention to the close parallelism between the amount of hemorrhage in the retina and the degree of anemia which is associated with the leukemia. This relation was noted by Borgeson and Wagener [1] in their excellent paper on leukemia.

DIAGNOSIS

In many cases it is possible for the ophthalmoscopist to make a positive diagnosis of leukemia without the assistance of the clinical data merely by the appearance of the fundus. In this series, the retinal changes were characteristic of leukemia in 2 cases before the changes in the blood and the clinical signs were sufficiently advanced to warrant a diagnosis. The ophthalmologic diagnosis was subsequently confirmed at a later admission to the hospital. Consequently, it does not behoove

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From the Department of Ophthalmology of Temple University Medical School.

Read before the Eye Section of the College of Physicians of Philadelphia, April 21, 1938.

1. Borgeson, E. J., and Wagener, H. P.: Changes in the Eye in Leukemia, Am. J. M. Sc. **177**:663-676 (May) 1929.
2. Goldback, J.: Leukemic Retinitis, Arch. Ophth. **10**:808-817 (Dec.) 1933.
3. Frank, J.: Leukemic Retinitis, M. J. Australia **1**:364 (March 23) 1935.

the ophthalmologist who finds a typical retinitis of leukemia to change his opinion if the clinician is unable to confirm the diagnosis. Twelve patients presented retinal changes which were sufficiently characteristic to permit a positive diagnosis of leukemia to be made. In 2 of the cases the diagnosis made on the basis of the fundal changes was blood dyscrasia, and in 1 case it was retinitis of anemia; in 2 of the cases no classification was made. The fundi were reported as normal in 5 cases.

The first and most frequently observed sign in the cases of leukemia in this series was a change in the appearance of the retinal veins. They became darker and fuller than normal. This was the most helpful sign in making the diagnosis. This observation is not in accord with that of R. Foster Moore,[4] who stated that the venous changes are the least significant. The retinal veins were recorded to be darker and fuller than normal in all except the 5 normal fundi. This sign almost invariably precedes the retinal hemorrhages and persists after they absorb during remissions. In the most advanced and typical forms the veins become dilated and tortuous and constricted at intervals, so as to resemble roughly a chain of sausages. In every case of bilateral involvement in which the retinal veins appear darker and fuller than normal, leukemia should be suspected, and a careful study of the fundi should be made to exclude such types of venous changes as are associated with anemia, polycythemia, congenital heart disease and congenitally large veins. This is not always easy or possible, but frequently one is rewarded for careful study by helpful differential points. Congenitally large veins are normal in color and quite tortuous and are usually matched by a similar tortuosity of the arteries in an otherwise normal fundus. In cases of anemia the rest of the fundus, including the disk, is pale. In the cases of leukemia the combination of dark full veins and pallor of the disks was not observed without other associated lesions. In cases of polycythemia there is a definite cyanotic color, and the arteries are also larger and darker than normal. In cases of congenital heart disease the arteries usually appear normal, and the veins, which are larger than normal, are extremely cyanotic. One should exclude a diagnosis of leukemia in cases in which one observes the combination of normal disks and arteries associated with relatively full and dark retinal veins. If, however, in addition to the changes just enumerated, one finds one or more retinal hemorrhages, the possibility of the condition being leukemia is enhanced materially. Retinal hemorrhages were observed in 13 of the 22 cases in this series at the first observation. In 2 cases localized areas of exudate without hemor-

4. Moore, R. F.: Medical Ophthalmology, ed. 2, Philadelphia, P. Blakiston's Son & Co., 1925, chap. 10, p. 104.

rhage were observed, hemorrhages occurring subsequently in the course of the disease. While the most typical hemorrhage of leukemia is a large round hemorrhage with a white center, this type is seldom observed in the earlier stages of the retinal changes. One may encounter almost any size or shape hemorrhage, with the possible exception of the small punctate hemorrhages so frequently observed in cases of diabetes. The degree of hemorrhage may vary from one small hemorrhage to almost complete infiltration of the retina. The amount of the hemorrhage seems to be proportional to the degree of anemia associated with the leukemia; hence in certain cases of leukemia a retinal picture is observed that is indistinguishable from the retinitis of anemia. In 2 of the cases the hemorrhage was so extensive that it simulated bilateral thrombosis of the central retinal vein. If, in addition to the foregoing changes, one observes a narrow white line on each side of the vein, the condition is true retinitis of leukemia in the limited sense, with perivenous diapedesis of the white blood cells. This perivenous infiltration does not in any way deform the vein, and this is helpful in differentiating the condition from periphlebitis. In 2 cases there was edema of the disk. Rarely one observes that the arteries become slightly fuller than normal and that the reflex stripe on the artery is accentuated.

PROGNOSIS

While the ultimate prognosis in cases of leukemia is almost invariably fatal, it is difficult to attempt to judge the immediate prognosis by the retinal picture. Occasionally the disease runs its complete course without the development of ocular signs. Consequently, a normal ocular picture does not necessarily render the immediate prognosis good. Usually in fatal cases an extensive hemorrhagic infiltration of the retina develops before death. However, it sometimes happens that a patient with maximum retinal involvement will enjoy a spontaneous remission both from the retinal and the clinical standpoint. Consequently extensive retinal hemorrhage, though usually a grave sign, does not necessarily have an immediate serious significance. Aside from these exceptions, however, one gets the clinical impression that there is a fairly close relation between the amount of retinal hemorrhage and the clinical condition of the patient. As a rule, when the patient's general condition gets worse more retinal hemorrhages occur, and when the patient improves clinically, the hemorrhages tend to absorb.

CORRELATION BETWEEN RETINAL HEMORRHAGE AND ANEMIA

In the analysis of these cases there was one observation which seems to be of clinical importance, that is, the close parallelism between the amount of anemia and the extent of the retinal hemorrhage. For

descriptive purposes, the cases were divided into four groups on the basis of the amount of retinal hemorrhage. In group 1 were included those cases in which there were from one to three retinal hemorrhages (fig. 1). Group 2 included those in which from about four to ten retinal hemorrhages occurred, while group 3 (fig. 2) consisted of those in which the hemorrhages were too numerous to count, and group 4 (fig. 3), those in which there was a maximum degree of retinal hemorrhage. The cases are arranged in order in figure 4, so that the ones in which the red cell counts were the lowest are at the top of the chart and those in which the red cell counts were the highest are at the

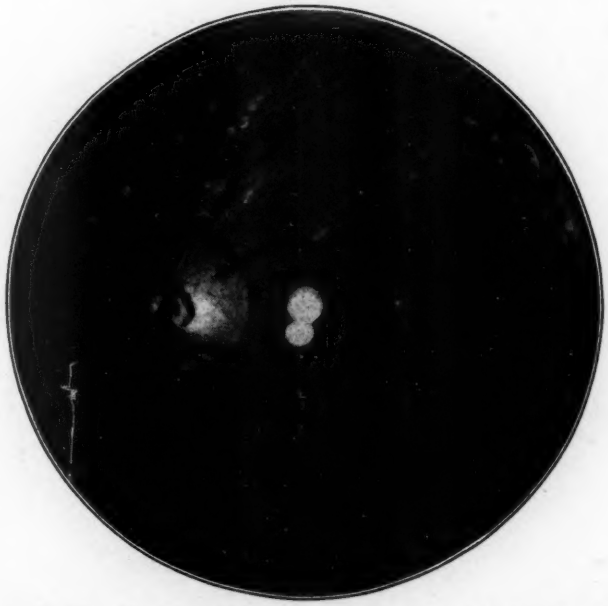

Fig. 1 (group 1).—Early retinal picture in a case of leukemia. The veins are slightly full and dark; the hemorrhage is near the normal disk; the arteries are normal, and exudate is seen above the macula. The rest of the white spots are high lights.

bottom. The degree of hemorrhage was plotted on the basis of the foregoing classification. It is immediately apparent that the cases in which the red cell counts were the lowest were the ones in which the most extensive degree of retinal hemorrhage occurred. Cases of anemia of a moderate degree are in general the ones in which the more moderate amount of retinal hemorrhage occurs, whereas those in which the red cell counts are relatively high are the ones in which no retinal hemorrhage occurs. This close correlation indicates that there must be a close relation between the amount of hemorrhage and the degree of

anemia. Further confirmation of this was furnished by examination of 3 patients who on first observation had no retinal hemorrhages and only a mild degree of anemia. Subsequently, it was observed that retinal

Fig. 2 (group 3).—Slight edema and pallor of the disk, as shown in the right and left eye of the same patient. The veins are dark, full and tortuous; the arteries are normal, and the hemorrhages are too numerous to count.

Fig. 3 (group 4).—Maximum degree of retinal hemorrhage with white centers, as shown in the right and left eyes of the same patient. Slight edema of the disk is present.

hemorrhages developed and that this development was associated with an appreciable decrease in the number of red blood cells. This decrease was out of proportion to the degree of the clinical hemorrhagic phe-

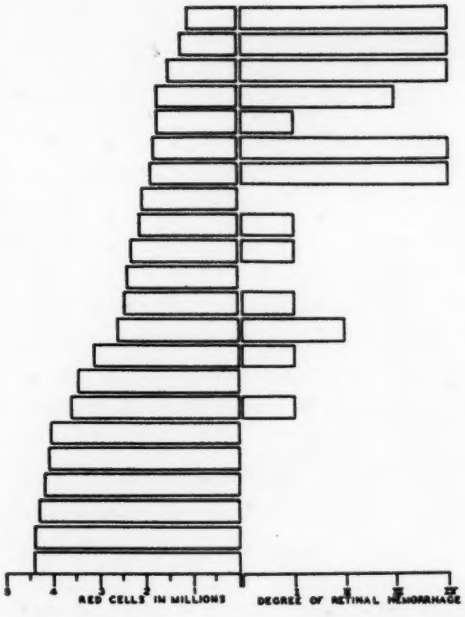

Fig. 4.—Correlation between anemia and retinal hemorrhage in cases of leukemia. The blocks to the left represent the red blood cells in millions in each of the 22 cases at the time of the first retinal examination. The adjacent blocks represent the amount of retinal hemorrhage. If there is no block adjacent to the red blood cell block, it indicates that there were no retinal hemorrhages. The extensive retinal hemorrhages occurred in the cases of severe anemia; the moderate degree of retinal hemorrhages occurred in the cases of moderate anemia, and no hemorrhages occurred in the cases of mild anemia.

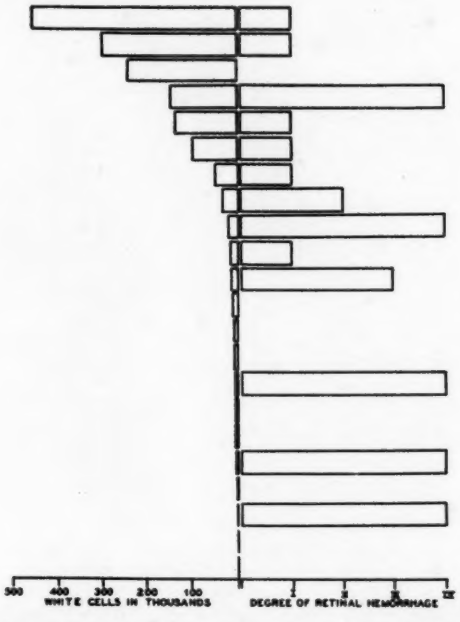

Fig. 5.—Comparison of the white cell count and retinal hemorrhage in cases of leukemia. The blocks on the left represent the white blood cell count in each of the 22 cases, and the blocks to the right represent the amount of retinal hemorrhage. There is no correlation between the leukocytosis and the amount of retinal hemorrhage.

nomenon. In this series of cases, it seemed to make no difference whether the leukemia was of the myelogenous or the lymphatic type. There is a similar but less consistent correlation between the hemoglobin and the retinal hemorrhages. Interestingly, in the case in which the red cell count was the lowest the ophthalmologic diagnosis was retinitis of anemia instead of leukemia. There is no such correlation between the white cell count and the amount of retinal hemorrhage, as pointed out by Goldback.[2] In figure 5 the same cases are arranged in the order of increasing white cell count, and it is apparent that there is no relation between the amount of retinal hemorrhage and the degree of leukocytosis.

CLINICAL IMPRESSION

Since there is such a close parallelism between the degree of anemia and the extent of the retinal hemorrhage as well as the clinical condition of patients with leukemia, it seems fair to conclude that evidence has been presented which suggests that therapeutic and investigative procedures could be more advantageously directed toward the factor of anemia instead of toward the leukocytosis in the unsolved problem of leukemia.

DISCUSSION

DR. FRANCIS HEED ADLER: I should like to ask Dr. Gibson if he has any idea what causes the white spots in the center of the hemorrhages in cases of leukemia. Textbooks usually say that they are due to the presence of accumulated white blood cells, but I have noticed them in cases of leukemia when the white cell count was not materially elevated.

DR. GLEN G. GIBSON: In observing these cases one gets the impression that the white centers are due to a collection of serum and hemorrhagic débris rather than to a nest of white blood cells surrounded by red blood cells. I regret that I have no microscopic confirmation of this impression.

BILATERAL CONGENITAL ECTOPIA LENTIS WITH ARACHNODACTYLY (MARFAN'S SYNDROME)

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The condition which bears Marfan's name is characterized by the following symptoms: lack of subcutaneous fat, poorly developed musculature, elongated extremities and curvature of the spine. In approximately half of the cases the following conditions are also found: congenital cardiac disease, congenitally dislocated lenses, miosis and high myopia. The condition occurs with equal frequency in both sexes and usually is not recognized until the third or the fourth year; often it is not diagnosed until adult life has been reached. It is a hereditary condition which can be transmitted by males and females alike and is considered by most authorities to be transmitted by changes in a varying number of chromosomes. If only chromosomes concerned with mesodermal development are involved, the ocular symptoms are absent (congenital mesodermal dystrophy); if chromosomes concerned with ectodermal development are also affected, the suspensory ligament of the lens and sometimes the dilator muscle of the iris are involved, resulting in dislocation of the lens and a rigid, contracted pupil (congenital ectodermal dystrophy).

The mesodermal changes are characteristic. The patient is tall, due to the long legs; the hands and feet are extremely long and thin; the body and face are thin, due to the lack of subcutaneous fat; the fingers are delicate and long ("spider digits"—arachnodactyly); there is scoliosis or kyphosis or both; the skull and teeth are long and narrow, and the bony palate is narrow with a high arch.

TERMINOLOGY

Some of the mesodermal changes were first described by Marfan [1] in 1896, and the name of arachnodactyly was suggested by Achard [2] in 1902. Since then variations in the syndrome, including the cardiac and ocular changes, have been observed. In 1924 Ormond and Wil-

1. Marfan, A. B.: Un cas de deformation congénitale des quartre membres plus prononcée aux extremities characterisée par l'allongement des os avec un certain degré d'amincissement, Bull. et mém. Soc. méd. d. hôp. de Paris **13**:220, 1896.

2. Achard, C.: Arachnodactylie, Bull. et mém. Soc. méd. d. hôp. de Paris **19**: 834, 1902.

liams [3] first described the ocular changes which occur in about half the cases of this syndrome. The only American ophthalmologists who have paid any particular attention to this condition are Lloyd [4] and Burch,[5] whose articles appeared in 1935, 1936 and 1937. Burch's article is accompanied by a complete chronologic bibliography, which it is unnecessary to repeat here.

Weve [6] reported on this condition in 1931 and added reports of 23 cases in 6 families from Holland. This is the first article to appear from that country. He placed all the signs on a mesodermal basis. Killmann [7] reported 7 cases in which the picture was typical and 1 in which it was not quite typical; the latter type he called a *forme fruste*. His experience was the same as that of all other investigators in that he found no evidence of syphilis, alcoholism or endocrine disturbance. Franceschetti [8] and Waardenburg [9] expressed the belief that the changes in the iris and lens are ectodermal in origin, and in 1 case he noted a coloboma of the lens.

Because of the congenital changes in the mesodermal and ectodermal tissues, with resultant dislocation of the lens, perhaps it would be advisable to coin a new name for the condition and call it dysmesectopia. The "dys" is for the dystrophy, the "mes" for mesodermal and the "ecto" for ectodermal; the "ecto" is also a part of the word ectopia (dislocation). If only the mesodermal tissue is involved, the name would be dysmesdactyly, the "dactyly" referring to the condition of the fingers, which is a prominent characteristic, and at the same time part of the term arachnodactyly, which has come into fairly common use, would be retained.

REPORT OF CASES

CASE 1.—M. H., a white man, single, aged 50, was first seen in Dr. Fletcher's clinic at the Manhattan Eye, Ear and Throat Hospital in December 1936, at which time he requested an examination for glasses. The vision in the right eye was found to be limited to the ability to count fingers at 5 feet (152 cm.); there was no improvement with glasses. The vision in the left eye was 20/200, which was

3. Ormond, A. W., and Williams, R. G.: Case of Arachnodactyly with Special Reference to Ocular Symptoms, Guy's Hosp. Rep. **74**:385, 1924.

4. Lloyd, R. I.: Arachnodactyly, Arch. Ophth. **13**:744 (May) 1935; A Second Group of Cases of Arachnodactyly, ibid. **17**:66 (Jan.) 1937.

5. Burch, F. E.: Association of Ectopia Lentis with Arachnodactyly, Arch. Ophth. **15**:645 (April) 1936.

6. Weve, H.: Ueber Arachnodactyly (Typus Marfan): Dystrophia Mesodermalis Congenita, Arch. f. Augenh. **104**:1, 1931.

7. Killmann: Angeborene doppelseitige Linsenektopie und Arachnodaktylie, Klin. Monatsbl. f. Augenh. **92**:335, 1934.

8. Franceschetti, A.: Marfanscher Symptomenkomplex und Coloboma lentis, Klin. Monatsbl. f. Augenh. **88**:686, 1932.

9. Waardenburg, P. J.: Das menschliche Auge und seine Erbanlagen, Haag, Nijhoff, 1932.

improved to 20/70 + by a + 1.00 D. sph. ⊃ + 2.00 D. cyl., axis 15. The right eye showed a deep anterior chamber, with the face of the iris somewhat concave, an extremely small pupil and marked iridodenesis. After several drops of homatropine hydrobromide and atropine were administered, only slight dilatation of the pupils was obtained, but it was sufficient to determine that in the right eye the lens was in the lower part of the vitreous and marked choroidal pigment changes were present throughout the fundus. The left eye also had a small pupil, and the face of the iris was concave below and convex above. Moderate iridodenesis was present. A cataractous lens could be seen dislocated up and in, and on movement

Appearance of the patient in case 1.

of the head in certain positions the edge of the lens could be moved partially out of the pupillary space. The patient was seen at regular intervals, and one year later the left lens was found entirely displaced from the pupillary space and lying in the vitreous below. The vision now was 20/30 with a +1.00 D. sph. ⊃ +2.00 D. cyl., axis 135. The fundus was clearly seen after slight dilation with homatropine hydrobromide. There were marked myopic changes, including a posterior staphyloma and choroidal atrophy.

The typical appearance of the hands and feet and the gaunt appearance due to the lack of subcutaneous fat are shown in *A* of the accompanying illustration. The extreme kyphosis is shown in *B*. In both *A* and *B* it will be noted that the

left lower extremity is more robust and more fully developed. This was due, I believe, to the kyphosis which resulted from excess weight being put on the left lower extremity.

According to the patient, his father and mother had no ocular trouble and wore glasses only for reading. He has two brothers, 1 of whom I examined and found to have normal eyes and no skeletal defects. The other brother is supposed to have normal eyes and has 2 children, one of whom is nearsighted.

CASE 2.—D. H., a 10 year old girl, was first brought to Dr. Fletcher's clinic at the age of 6 years because of poor vision. When examined at the first visit she was found to have long hands and feet and looked emaciated. No subcutaneous fat was present, and there was marked lordosis of the spine. Her teeth were long and thin, and her palate was high and quite arched. The lenses were both dislocated, and the irides were tremulous. The pupils were small but were easily dilated with homatropine hydrobromide. The fundi showed no abnormalities. At the time of writing, four years later, both lenses are completely disclocated and lie in the vitreous below. With a + 9 D. sph. vision of approximately 20/50 is obtained for each eye, but it is difficult to examine the child as she is not cooperative. Her father and mother and maternal grandmother have normal eyes and no skeletal defects. The child has no brothers or sisters. A request for a photograph was emphatically refused by the patient.

COMMENT

There has been a good deal of discussion about the condition of the dilator muscle of the iris in cases of dysmesectopia. Some say that the muscle is rigid and others are as certain that it is absent. In 1 of my cases (2) dilatation was easily obtained with homatropine hydrobromide, showing that the dilator muscle was not rigid, and moderate dilatation was obtained with cocaine hydrochloride, pointing to the presence of some dilator muscle fibers. In the other case (1) only a slight increase in the size of the pupil, 2 mm., was obtained with either homatropine hydrobromide or cocaine hydrochloride, but the dilatation was definite. I think that a conservative opinion would be that in some cases of dysmesectopia not only mesodermal structures are affected but ectodermal structures, such as the zonular fibers of the lens or the dilator muscle of the iris or both, may also be involved. This is what Vogt [10] suggested, and many others agree that it seems correct in the light of the present knowledge.

SUMMARY

Two cases of congenital mesodermal and ectodermal dystrophy with arachnodactyly and bilateral congenital ectopia lentis are reported, and a résumé of the various signs and symptoms is given. The terminology is discussed, and it is suggested that the term dysmesdactyly be adopted when the chromosomes concerned with mesodermal tissue are involved. When the ectodermal tissue is also affected, the term dysmesectopia is suggested.

10. Vogt, A.: Marfan's Syndrome, Klin. Monatsbl. f. Augenh. **87**:258. 1931.

NATURE AND MANAGEMENT OF THE HETEROPHORIAS

J. T. MAXWELL, M.D.
OMAHA

At birth the eyeballs assume a position controlled by basic innervation which is present before environmental influences have become effective. This innervation is innate and has been called tonic innervation. At birth vision is not fully developed, nor is the relation between the two eyes developed. As growth proceeds, fixation, fusion, coordination, convergence and accommodation develop, become interrelated and assume their various roles in the positioning of the eyes. When the higher centers of the brain are inhibited, as in sleep, deep narcosis or coma, the eyes may assume a position which is irrelevant to that in effect when the nervous system is functioning normally. In fact, it seems from the positions of the eyeballs under the conditions named that the eyes are more or less set adrift to wander with little or no innervational control. With the return to normalcy, a flow of properly distributed innervation to the ocular muscles is resumed. In discourses on psychophysiologic mechanisms, the term position of rest is frequently used. The question arises as to what may be called rest. Shall the term be used when fusion alone is eliminated or when other stimuli which influence the position of the visual axes are rendered inactive?

TONIC INNERVATION

When a dissociation method, such as the screen test or the Maddox rod, is used to test the muscle balance for distant vision (assuming emmetropia), only the tonic innervation is in effect, because fusion is eliminated, and if the eyes are emmetropic, no accommodation is active. Under dissociation, there may be either orthophoria or heterophoria (exophoria, esophoria, hyperphoria or cyclophoria).

Exophoria may be defined as that position of the eyes, relative to the distance at which the test is made, in which the visual axes, under dissociation, do not meet before reaching the object of fixation. Esophoria is that position of the eyes in which the visual axes, under dissociation, meet between the eyes and the object of fixation. Hyperphoria and cyclophoria are likewise positions of the eyeballs controlled by the innate tonic innervation when fusion is made artificially impossible. In other words, heterophorias are examples of improper distribution of

tonic innervation. It is the function of the fusion faculty to correct these anomalies by keeping the eyes in a position that will insure single binocular vision. The adequacy of this mechanism depends on the degree of heterophoria and the extent of the fusional amplitude. Exophoria or esophoria for distance vision of over 1 or 2 prism diopters which is not caused by accommodative convergence would be an example of an anomaly of tonic innervation.

When the relation between accommodation and convergence is well developed, esophoria may result from accommodative convergence prompted by the effort of the accommodation to overcome an existing hyperopia. The same condition may result from an overcorrection of myopia. This would not be an anomaly of tonic innervation. An esophoria of this type would disappear under the influence of a correcting sphere. The adjustment might not occur at once, however, owing to the possible formation of habit.

The detection and measurement of anomalies of tonic innervation are accomplished by dissociation methods at 6 meters. In the presence of such an anomaly the power of the fusion faculty must be measured by prism convergence or prism divergence (duction tests) in order to ascertain the adequacy of the correcting force in overcoming the existing phoria.

Heterophoria is not in itself necessarily of clinical significance. It is the ratio of the correcting power of the fusion faculty to the heterophoria which is the deciding factor. If the power of the fusion faculty is low in proportion to the amount of heterophoria present, single binocular vision will either be maintained under stress (eyestrain) or, if the proportional strength of the fusion faculty to the heterophoria is too low to produce single binocular vision, strabismus will result. Exophoria of 5 prism diopters for distance vision would in all probability give no trouble if the eyes were able to overcome 10 or more prism diopters base out (adduction) and retain single binocular vision; whereas, if the positive fusional convergence were much less, the exophoria for distance vision might well be expected to produce symptoms. It is the relation of the demand (heterophoria) to the available innervational supply indicated by the duction tests which decides the issue.

By long persistent orthoptic training it is often possible to reduce a phoria, but the process is not an easy one. In fact, if some simple method could be developed whereby a phoria could be readily changed, the greatest problem of ocular muscle imbalance would be solved.

In contrast to the relative stability of the distribution of tonic innervation, the power of the fusion faculty may vary greatly, not only with the condition of the general system but as the result of orthoptic training and other influences.

FUSIONAL RESERVE

If a heterophoria exists, the fusion faculty should be able to maintain single binocular vision, and in addition there should be a reserve power, measured by prisms. As an analogy, one might say that if a man expected to carry a weight of 50 pounds (23 Kg.) for a city block, he should be able to lift a considerably greater load. If the fusion faculty were just barely able to compensate a phoria (no reserve) and produce single binocular vision, it would be unreasonable to suppose that the condition would be comfortable over an extended period of time.

When both eyes fixate an object (single binocular vision) and prisms, base in or base out, are interposed, the images begin to move off the fovea, and a reflex is set up which will, within certain limitations, replace the images on the fovea and maintain fusion. The important aspect of the situation is the limitations. When prisms are placed base out before the eyes, they will, in order to retain single vision, converge to a certain extent only, beyond which limit the addition of further prism power will produce either diplopia or suppression of vision in one eye. The same is true of divergence produced by base in prisms, except that the out-turning of the eyes is usually less in amount than the inturning.

It can easily be shown that the angular distance which the eyes may be turned by prism power while maintaining fusion is not a muscular limitation, because as soon as fusion is lost either eye will turn farther. It is evidently the association factor that imposes the limitations. The degree to which the fusion faculty is developed is apparently the controlling factor.

A strain is produced under prism action in proportion to the strength of the prisms used. Under such conditions a load is thrown on the fusion faculty in an effort to retain single binocular vision. Thus, by the use of prisms, the power of the fusion faculty is measured. That amount of prism power which can be overcome, in excess of that which is normally in use, has been called the "fusional reserve." Comfortable vision depends largely on the relation of the amount of fusion power in use to that in reserve. Experience has shown that eyestrain is likely to occur if over one half of the total fusion power is in use. Such a rule will have many exceptions and be subject to individual variation; nevertheless, it is a basis on which to work.

When, by orthoptic training, an attempt is made to produce a comfortable condition by building up the fusion faculty to compensate for a high degree of heterophoria, an acquired reflex is being artificially increased to compensate for an improper (innate) tonic innervation. It usually happens that even though the fusion faculty has been increased by training so that a heterophoria may be carried in comfort, the tonic innervation (heterophoria) will remain unchanged. I wish to emphasize particularly the fact that tonic innervation is stable and difficult to change.

INNERVATION AT THE READING DISTANCE

At the reading distance there must be considered not only the tonic innervation and the fusion faculty which are present for distance but the influence of accommodation and the proximity of the object of regard. In order to see near objects clearly and singly, the visual axes must converge, and accommodation must be in effect. Therefore, accommodation and convergence become associated, although either may act without the other. In the presence of uncorrected hyperopia, accommodation must be exerted for distant vision, and it can easily be shown with the dissociation tests that convergence is induced by this accommodation. Therefore, esophoria is likely to be present for distance vision in cases of uncorrected hyperopia. When the necessary plus lenses are added, accommodative esophoria will disappear. At the reading distance accommodative convergence is usually present. The difference between the exophoria for near vision and the total amount of convergence necessary to maintain single binocular vision for near objects indicates the amount of accommodative convergence which is in effect under dissociation. In persons with myopia, who accommodate little, if any, the exophoria for near vision will usually be high, because accommodation and convergence have not become as closely associated in such persons. Experience has shown that accommodative convergence for near vision in perfectly normal subjects may vary from 16 prism diopters (the total amount of convergence at 38 cm.) to none at all. It is therefore evident that accommodative convergence (calculated by the exophoria for near vision) is not necessarily an important factor of comfort for near vision but is simply indicative of the established relation between accommodation and convergence.

Convergence may be observed when an object approaches the eyes and may also be demonstrated by covering one eye with a card while a test object is brought near the other eye. The eye behind the screen, in most instances, will turn inward. There must be a stimulus to prompt a response. The question arises as to the sources of innervation which produce convergence at the reading distance. In order to differentiate these reflexes, they must be dissociated as in the distance tests. Fusion may be prevented by means of the Maddox rod, or the double prism may be used, or a single vertical displacing prism may be placed over one eye.

In this demonstration, the test object, such as a capital E 3 or 4 mm. high in the center of a card about 5 inches square, is placed on the arm of the phorometer at a distance of 38 cm. from the eyes of the subject. A 6 prism diopter base down over one eye will usually be sufficient to produce diplopia and cause one image to appear on a higher level than the other. If the two images are vertically alined, it is evident that

the same amount of convergence is in effect when fusion is eliminated as when single binocular vision is present. However, the images will not often be exactly one above the other. With the base down prism over the right eye, the upper image will most often appear to the left (less frequently to the right) of the lower image, thus indicating that the visual axes have, under dissociation, failed to converge the full 16 prism diopters. The amount of exophoria present for near vision is measured by the prism power base in which is necessary to aline the images vertically. The difference between the total amount of required convergence (16 prism diopters) and the exophoria for near vision (assuming orthophoria for distant vision) must be attributed to the influence of accommodation and the proximity of the object. For example, if there is an exophoria for near vision of 6 prism diopters and a total convergence of 16 prism diopters, there will remain 10 prism diopters of convergence because of the accommodation and the proximity of the object.

For the purpose of further analyzing convergence under dissociation at the reading distance, a pair of plus 2.75 D. spheres are added to relax accommodation. Any convergence present under these conditions cannot be due to either fusion or accommodation, since each eye sees a separate image from the effect of the prism (dissociation) and the spheres eliminate accommodation. It is reasonable to assume, therefore, that it is the psychic effect of nearness that induces the remaining convergence. For example, if after the plus spheres are added the images separate to such an extent that the exophoria for near vision is increased to 12 prism diopters, there is still left 4 prism diopters (16 prism diopters — 12 prism diopters) of convergence, which is attributed to the psychic effect of nearness of the object (promixal convergence).

With fusion eliminated, the visual axes converge only 10 prism diopters. When accommodation is inhibited by the plus spheres, 6 prism diopters more of convergence disappear, leaving the final 4 prism diopters to be attributed to proximity. Thus, it is seen that the factors which may produce convergence for near vision can be divided into fusional, accommodative and proximal. In practice, the proximal convergence and the accommodative convergence may be treated as one. They will be so considered in the following discussions.

At birth there is no relation present between accommodation and convergence. The accommodative convergence reflex is the result of a learning process. However, the degree of the development has little, if any, bearing on ocular efficiency or comfort. Proof of this premise is found in everyday practice, in which the ophthalmologist constantly encounters persons with low accommodative convergence who are perfectly comfortable. This reflex is simply the result of an association that has been present from infancy, when accommodation was necessary

to enable the person to see clearly and convergence became necessary to enable him to see singly. This being the case, it is not advisable to use the exophoria for near vision from which to compute convergence efficiency at the reading distance (38 cm.), since the exophoria for near vision merely determines the amount of accommodative convergence.

Most difficulties from ocular muscle imbalance arise from deficient positive fusional reserves at the reading distance. When the condition of balance at the reading distance is considered, it must be remembered that 16 prism diopters of positive convergence (orthophoria at distance) is necessary for single binocular vision at 38 cm. The eyes should be able to converge at least double the amount needed for fixation. Therefore, if the prism convergence (adduction) for near vision is less than 16 prism diopters, treatment may be needed.

MANAGEMENT OF THE HETEROPHORIAS

There are many persons with ocular muscle imbalance for whom orthoptic training is the only treatment indicated, although it is frequently advisable to use both training and prisms. The use of prisms without orthoptic training is indicated for the correction of hyperphoria and for lateral imbalances on occasions when exercises are impracticable.

For deficiencies of the fusional power in lateral imbalance, orthoptic training is direct in its application, since a weak fusional power is strengthened by training. In the tonic forms of ocular imbalance (phorias), the treatment is indirect in that strength (fusion) is built up in one faculty as compensation for a faulty distribution of another faculty (tonic innervation). By this means a comfortable condition may be produced, but the tests in most instances will show that the exophoria or the esophoria still remains, indicating that the tonic innervation is unchanged.

The methods available for the treatment of ocular muscle imbalance when single binocular vision exists consist of orthoptic training, the prescription of prisms for constant or temporary wear or a combination of both types of treatment. The indications for treatment are the same, whether orthoptic training or prisms are to be used.

Prisms incorporated in spectacle lenses are prescribed for the correction of heterophoria when for any reason orthoptic training cannot be given or when both methods of treatment are used. When the two are used together, the prisms are prescribed for immediate relief with the hope that they may eventually be discarded when orthoptic training has been successfully used.

When prisms are placed base in (fusion present), the eyes assume a position whereby the images are placed on the maculas with the visual

axes relatively divergent in respect to the fixation point; that is to say, base in prisms lessen the amount of convergence. On the other hand, base out prisms produce the opposite effect, so that the amount of convergence necessary for single binocular vision is increased above that normally required. It is apparent that the application of prisms is not curative in effect; thus, prisms are similar to crutches in this respect, but they are often necessary.

Prisms are prescribed chiefly for insufficiency of convergence and for vertical imbalance. A person who is orthophoric for distance vision must converge approximately 16 prism diopters in order to attain single binocular vision at 38 cm. (15 inches). Unless a reserve of converging power equal to at least the amount in use can be overcome without producing diplopia, it may be assumed that an existing eyestrain not otherwise accounted for is the result of insufficient positive fusional amplitude. If exophoria for distance vision is present, more than 16 prism diopters of convergence is necessary to fixate at 38 cm.; if esophoria for distance vision is present, the amount of convergence in force to maintain single binocular vision for near objects would be proportionately less.

The question immediately arises as to how much prism power shall be prescribed (orthoptic training not available) to produce comfortable use of the eyes for near work. In attempting to establish a schedule, one is confronted with the difficulty which always arises when physiologic processes are reduced to mathematical formulas. The following procedure is suggested:

If single binocular vision is barely maintained at 38 cm., that is, if diplopia appears as soon as a small amount of base out prism power is placed before the eyes, the maximum amount of base in prism to be prescribed for the first trial lens should not exceed 2 prism diopters for each eye. When some fusional amplitude is present but not equal to double the amount required for single binocular vision at the given distance, the strength of prism power will vary in proportion to the deficiency. For example, if the patient is orthophoric for distance vision and can overcome only 8 prism diopters base out at 38 cm., he might be said to have a deficiency of 8 prism diopters; since he should have a minimum of 16 prism diopters of reserve converging power but has only 8 prism diopters. If one assumes that a total of 4 prism diopters is the maximum that can be worn with comfort, a total of 2 prism diopters would be prescribed, 1 prism diopter base in before each eye. For slight deficiencies, the required effect can often be attained by decentration of the lenses without the necessity of grinding the prisms.

Attention must again be called to the fact that no rule can fit all cases and that any set rule is definitely empiric. If the first attempt at relieving the symptoms does not prove satisfactory, a modification of

the prescription must be made according to the indications and the judgment of the refractionist. The discomfort produced by strong prisms is no doubt in part due to prism aberration.

A prominent group of persons who require prismatic help are those with presbyopia who have insufficient converging power for near vision. For such persons prisms may be incorporated in the near correction, either as glasses for reading only or in the segments of bifocal lenses. It is a good plan to select an occasion when the patient has time to read in the reception room for an hour or two with trial lenses in order to determine the effect of the intended prescription. The person without presbyopia who is emmetropic and has insufficient convergence is confronted with the necessity of using prisms only for near work if he refuses orthoptic training.

The prescribing of prisms for the correction of esophoria is of doubtful value. This is especially true of high esophoria for near vision, which is usually of systemic origin. Yet many persons with esophoria are often uncomfortable, and base out prisms are in some instances gratefully accepted. Base out prisms should be prescribed with the patient's full knowledge that they are not of certain value, so that too much will not be expected.

Prisms in spectacles are used extensively for the relief of hyperphoria, since orthoptic training for vertical imbalance seems to be of little value. The amount of prism power prescribed should always be less than the amount of the phoria and depends on the prism power which the fusion faculty is able to overcome in the opposite direction (infraduction). It is suggested as a first trial that a prism be prescribed equal in power to one half of the demonstrable phoria, if the infraduction is less than the hyperphoria. If the vertical duction is equal to, or greater than, the phoria, proportionately less prism power should be prescribed. Errors of less than 1 prism diopter of hyperphoria are seldom considered of sufficient importance to require correction. Hyperphorias of high degree, especially when they are of sudden onset, are usually of toxic origin, and, on account of their fluctuating character, respond uncertainly to correction with prisms.

Many practitioners feel that the prescribing of prisms for constant use is questionable and fraught with the possible danger of increasing the error. With this thought in mind, consideration will be given to just what prisms in spectacle lenses accomplish. It is evident that prisms allow single binocular vision with the visual axes intersecting either nearer or beyond the object of regard. For example, if base in prisms are prescribed, less positive convergence is necessary than would otherwise be the case. If the fusional convergence is later required to do full duty by the removal of the prisms, symptoms of fatigue might arise. In other words, the patient would feel the effort more than before relief

had been afforded. In this respect the condition might be said to be made worse, at least the subject might so contend. On the other hand, the basic situation, that is, the exophoria, would in all probability not be altered, and from that standpoint the condition would not be increased unless the distribution of tonic innervation (phoria) had changed.

MEASUREMENT OF CONVERGENCE EFFICIENCY

In conclusion, I wish to call particular attention to the fact that convergence efficiency for near work must be determined by prism convergence with the test card placed at the reading distance. This procedure has a decided advantage over the method which consists of causing a test object to approach the nose while the point at which diplopia occurs or when one eye deviates is noted. In the "approach-the-nose" method, factors are introduced which are not in effect with fixation at from 38 to 40 cm.

In the first place, excessive accommodation is required, which may act as a further stimulus for convergence. In the second place, when the near point of accommodation has somewhat receded, as in cases of presbyopia, the resulting blurred image inhibits fusion and produces diplopia sooner than would otherwise be the case. This factor varies with the age of the patient. In the third place, the image on the retina becomes larger as the distance between the object and the nodal point of the eye shortens, introducing another factor not present in the use of the eyes at the ordinary reading distance. It is also possible that the increased proximity of the object may act as an additional stimulus to convergence. Objection to the near point method of measuring convergence lies in the fact that it is difficult to analyze and is not readily adaptable to a scale of measurement which can be associated with the converging power for distance vision or with the procedure for treatment. It seems that no correlation has been established between the near point of convergence for different ages and the necessary prism power used as a corrective measure. Above all, it is common to find persons having difficulty in reading, despite the fact that the near point of convergence is quite close to the eyes; yet when convergence is induced at from 38 to 40 cm. by means of base out prisms, it is found deficient. The last objection abrogates this test and relegates it to the discard as far as determining convergence efficiency is concerned.

OPTOCHIASMIC ARACHNOIDITIS

IMPORTANCE OF A MIXED TYPE OF ATROPHY OF THE OPTIC NERVE AS A DIAGNOSTIC SIGN

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In September 1926 a white man 25 years of age presented himself with a history of failing vision of six months' duration. He had suffered an occasional severe frontal headache ever since he had received an injury to the back of his head from a fall seven years prior to admission to the hospital. Roentgenograms of the skull showed no lesion; an examination of the sinuses revealed no pathologic process. The Wassermann reactions of the blood and spinal fluid were negative. Both optic nerve heads showed a mixed type of atrophy (visible lamina; sharply defined margin, and markedly reduced vessels, especially arteries). The fields of vision suggested a suprasellar lesion; the history of injury, a chronic serous meningitis or arachnoiditis involving the chiasm. The patient gave his consent for an exploratory craniotomy, which was performed on March 7, 1927, by Dr. George J. Heuer. The pia-arachnoid over the whole area of the brain exposed was much thickened and grayish white. Beneath it was a large amount of cerebrospinal fluid, which escaped in quantities when the tough pia-arachnoid was torn with a needle. Further investigation revealed a mass of adhesions about the anterior part of the chiasm, continuing forward around the optic nerve. The cisterna chiasmatis was distended with fluid, and its wall was thickened and opaque. The cisterna was broken into between the tuberculum sellae and the chiasm, and a large amount of clear cerebrospinal fluid escaped. As much of the wall of the cistern as possible was snipped away with scissors. With a blunt dissector, the adhesions around the optic nerves were separated so as to free the nerves as much as possible. They appeared attenuated and grayish. After these manipulations were completed, it was possible to elevate the chiasm, so that one could look into the sella turcica and see the diaphragm sellae. Nothing suggestive of a tumor was seen about the chiasm. The patient had a smooth convalescence. A month later the central scotomas and enlarged blindspots were smaller, and the headaches had disappeared. The vision progressively improved from ability to count fingers at 5 feet (152 cm.) with each eye prior to the operation to 20/30 + in each eye one and one-half years later. A recent letter stated that the patient's vision has remained good and his health excellent. (This case was reported by Heuer and myself in 1931.)

This case is considered in detail because it was the first of its kind encountered by me and especially because most of the characteristics were present which have since been established as a disease entity. Chronic arachnoiditis, or arachnitis affecting the brain and spinal cord, is well known. The generalized form was first described by Quincke in 1893 and the localized form by Schlesinger in 1898. Since this time comprehensive reviews of the whole subject have been published by

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many authors, more recently by Craig, Spurling, Horrax and Stookey. The most important contributions on this subject, however, have recently been published by French authors, notably the monograph by Bollack, David and Puech. Because this paper is limited to a discussion of arachnoiditis producing the chiasmic syndrome, it is not pertinent to consider arachnoiditis affecting other parts of the central nervous system, except to mention the etiologic and pathologic factors which are probably common to all types of this disorder.

The cause of chronic arachnoiditis is not definitely established. Heuer expressed the belief that when an etiologic factor is present, it falls into one of two groups: an antecedent trauma (fracture of the skull or spine) or an infection. The infections are of two kinds: an infection of the meninges or brain (encephalitis, meningoencephalitis or leptomeningoencephalitis) or an infection of an area adjacent to the brain (the middle ear, the mastoid or the various sinuses). Most writers on the subject have noted the same etiologic factors. The pathologic process has been described by many authors who are in general agreement regarding the process and modus operandi. A thickened arachnoid is connected to the adjacent nerve tissue by fibrous bands, which vary in size from small discrete lines of adhesions to dense processes extending over a large area. The adhesions may form pockets and block the free flow of spinal fluid, producing the so-called arachnoid cysts. Localized symptoms develop, depending on the location of these bands and cysts, and produce what the older writers called pseudotumors.

The pathologic process affecting the chiasm is similar to that affecting the arachnoid. The clinical signs point to a restricted lesion in the chiasmic region. However, it has been observed by all neurosurgeons that the entire adjacent structure of the brain is involved by a more or less generalized process. It is therefore probable that if it were not for the accumulation of spinal fluid around the chiasma and the pressure symptoms produced thereby, the disease might remain silent and unrecognized. If this is true, it is logical to believe that there are many cases of chronic arachnoiditis in which the disease has never led to symptoms or distress. In the article by Heuer and myself this point was commented on as follows:

> It has been our experience generally that chronic arachnoiditis is a fairly widespread disease regardless of the fact that the local symptoms produced by it may be referred to the cerebellum, the motor cortex, the chiasmal region or a certain level of the spinal cord; in other words, localized arachnoiditis is, in our experience, identified by symptomatology rather than pathology.

In 1929 Manual Balado, of Buenos Aires, reported the case of a man aged 35 who complained of progressive visual failure of six months' duration. At the time the patient was seen his vision was reduced to

perception of shadows and light. Both disks showed primary atrophy. The optic chiasm was exposed, and an area of circumscribed inflammation of the arachnoid was found surrounding it. A piece of the arachnoid was removed, and histologic study showed some proliferation of endothelial cells and small calcareous nodules. During the months after the operation the vision steadily improved. This is the first case of chiasmic arachnoiditis on record, and Arce has proposed, with some justice, I believe, that the chiasmic syndrome produced by this disease be given Balado's name. However, Cushing and Eisenhardt, in discussing meningiomas arising from the tuberculum sellae, apparently recognized and reported this disease entity nine months before Balado. They said:

> Though this chronic cisternal arachnoiditis, apart from a postmortem examination, must always be a questionable diagnosis, cases nevertheless are encountered in which, due probably to a previous inflammation, a local accumulation of fluid trapped in a thickened arachnoid membrane may give symptoms of tumor. . . . In a few cases, however, in which a suprasellar lesion was suspected on the grounds of the syndrome under discussion, nothing was found but a local collection of slightly xanthochromic fluid, the evacuation of which led to a subsidence of symptoms.

It is unfortunate that reports of these few cases were not collected and published under a proper isolated title, since then there would be no question of priority. It has been my good fortune and privilege to observe and study 8 patients with arachnoiditis affecting the optic chiasm. Seven of them were operated on, and the diagnosis was verified at the time of the operation and by the subsequent progress of the patient. The eighth patient was not operated on, but his progress, and especially the changes in the visual fields, justify me, I believe, in including this case in my series. The visual fields first showed bilateral central scotoma, then bitemporal contraction, with progressive loss of the visual field practically to zero, followed by recovery in the reverse order. The patient was treated with injections of a foreign protein (typhoid vaccine intravenously), large doses of sodium salicylate (from 100 to 150 grains [6.5 to 9.7 Gm.] per rectum) and frequent spinal tapping. Later, 15 grains (0.97 Gm.) of sodium iodide was given intravenously daily for a course of fifteen injections.

A study of my cases and of those reported in the available literature (a total of 134 in which the diagnosis was verified at operation) has allowed me to draw certain conclusions. The condition is a disease of young adults, over 80 per cent of the cases occurring in persons under the age of 40. Seven cases in which the patients were under 10 have been reported. The condition predominates slightly in the male. The vision in one eye may be affected long before the second eye is involved; in 1 case there was an interval of ten years (Vincent). Once the disease

begins, however, the loss of vision is likely to be rapid. Occasionally slight remissions are observed. The earliest symptoms are most frequently those of acute retrobulbar optic neuritis, and there is almost always a central or cecocentral scotoma found in the field of vision. Bitemporal, binasal or homonymous hemianopia are frequently associated with the scotoma. Several cases of a basofrontal syndrome have been reported, with papilledema on one side and atrophy of the optic nerve secondary to papilledema on the other. Several combinations of signs may occur (Craig-Lillie): (1) unilateral central scotoma with normal fundi; (2) unilateral central scotoma and pallor of the disk but normal conditions in the other eye; (3) bilateral central scotoma with pallor of both disks; (4) bilateral central scotoma with choked disks; (5) unilateral amaurosis with simple atrophy of the optic nerve and choked disk in the opposite eye, sometimes associated with a central scotoma; (6) central scotoma and various alterations of the peripheral fields due to secondary contraction resulting from choked disks, and (7) bilateral amaurosis with any of the foregoing changes in the fundus.

It is my personal experience and belief that the appearance of atrophy of the disk which lies between the primary and the secondary type of atrophy is highly suggestive of arachnoiditis involving the chiasm. It would be unwise to be bold enough to assert that this is a pathognomonic sign, yet one is struck by the frequency of cases in which the outline of the disk is sharply defined, the lamina cribrosa is visible, but the caliber of the vessels is markedly reduced. I have observed this sign even when there has been a preceding papilledema of 2 diopters with hemorrhages of the retina. On the other hand, the case reports in the literature, while not complete in regard to this point, are highly suggestive that many of the patients showed the mixed type of atrophy of the optic nerve characterized by a blurred margin of the disk, normal-sized vessels and the presence or absence of the lamina cribrosa associated with either complete or sector atrophy of the nerve head.

Puech and Mahoudeau considered that the early and rapid simultaneous papillary edema and pallor of the nerve head coinciding with an important lowering of the visual acuity are characteristic of optochiasmic arachnoiditis. In other words, a study of the cases reported in the literature, associated with the clinical findings in my own experience, suggests that the involvement of the optic nerve is a combination of papilledema and simple atrophy. Either one or the other may predominate, and the resultant atrophy will take on a mixed character.

An analysis of the 129 cases studied by Bollack, David and Puech brings out the following facts regarding the nerve head: papillary atrophy with a sharp outline, of the primary type, 38 per cent of the cases; atrophy with a blurred outline, 15 per cent; papillary stasis, 10 per cent; partial atrophy limited to the temporal segment, 7 per cent;

simple hyperemia, 7 per cent; partial horizontal atrophy, 4 per cent; normal disks, 10 per cent, and disks difficult of interpretation, 8 per cent. However, the so-called primary type of atrophy of the optic nerve reported in 38 per cent of their cases was apparently not described in a thorough fashion, which would include a description of the outline, the elevation and the color of the disk, the visibility of the lamina cribrosa and the size and shape of the blood vessels. A history of previous inflammatory illnesses is found in many cases. These include syphilis, sinusitis, infected tonsils, abscessed teeth, whooping cough, encephalitis and especially influenza. In only a few of the analyzed cases was there a definite history of trauma, and in none of these was the injury presumably fracture of the skull. One is inclined to think, therefore, that trauma plays little or no part in producing this particular syndrome. Severe headache but rarely vomiting accompanies visual loss. Nystagmus has been observed in a few cases. Polyuria and polydipsia are occasionally complained of, indicating irritation if not actual inflammation of the hypothalamic area. Studies of the visual fields are of paramount importance, not only prior to surgical intervention but also afterward, since it frequently happens that recovery of the field of vision progresses by the same stages as the loss, but in reverse order. There is, however, nothing typical of the defects in the visual field to help much in diagnosing this condition. Many varieties of defects in the peripheral visual field are described, but a central scotoma associated with a hemianopic defect of the peripheral visual field, especially if bitemporal, is most helpful and suggestive. The central scotoma precedes the defect in the peripheral field as a rule. According to Rollet, the characteristic alterations of the visual field in cases of arachnoiditis are as follows:

> . . . marked disturbance of the superiolateral quadrant, less marked in the superior internal quadrant; relative integrity of the externoinferior quadrant. If the lesions are very accentuated, an inferiointernal island in the form of a crescent or L-shaped sector embracing the point of fixation in its concavity may persist. In tumors, it is frequently the superior internal quadrant which is the last to be preserved. These alterations are explained by the predominance of lesions on the posterior and inferior surface of the chiasma, these areas being the most exposed to meningeal infections.

A frequent observation is the remission of visual loss in the course of the disease. The ocular symptoms improve momentarily, and one finds spontaneous amelioration of visual acuity, but at the end of some time there is a new attack and loss of vision. This, of course, is a frequent finding in cases of multiple sclerosis.

In most cases the cerebrospinal fluid is found to be normal. In only a few has there been increased pressure. Likewise, one rarely encounters general neurologic signs. Roentgenograms of the region of the

sella turcica show no destruction. In a few cases calcified areas were observed, leading to an erroneous diagnosis of a tumor of Rathke's pouch. Ventriculography is of no value in diagnosis.

The differential diagnosis offers many difficulties. Cushing classified the ocular syndromes produced at or near the optic chiasm as follows: 1. Meningiomas, with a parasellar rather than a suprasellar point of origin. These generally occur in persons who complain only of impairment of vision; the visual fields become bitemporally constricted. 2. Adenomas of the pituitary gland. Roentgenograms showing enlargement of the sella turcica and signs of hypophysial dysfunction are the basis for diagnosis. 3. Congenital tumors arising from the craniopharyngeal pouch. These usually manifest themselves early in life, and roentgenograms reveal shadows caused by deposits of calcium in the wall of the cyst. 4. Gliomas arising from the chiasm or the third ventricle. These are relatively rare and often have an associated anterior pouching of the sella turcica, as shown roentgenographically. 5. Syphilitic meningitis, as revealed by a Wassermann test of the spinal fluid. 6. Aneurysms situated around the chiasm. These seldom have an audible bruit. The roentgenographic picture is often pathognomonic.

Since the earliest sign in all cases is retrobulbar neuritis, all causes of this condition must be considered in arriving at the diagnosis. The difficulties are enormous in the present state of knowledge, and several hours could be consumed in discussing retrobulbar optic neuritis from every angle.

Retrobulbar neuritis has been defined by Fralick as a disease of the visual pathways characterized by a selective affinity for the papillomacular fibers. It is a clinical entity irrespective of the ophthalmoscopic appearance of the optic disk, which may vary from normal to an extreme degree of swelling.

There may be gradual or rapid loss of central vision over a variable period of time, followed in most instances by a definite improvement of variable amount, regardless of the actual cause or the therapeutic measures used. The loss of vision may be partial or relative, complete or absolute, unilateral or bilateral and transitory several times a day. Headache or dull pain in the orbit is elicited on pressing or moving the eyes. This is not a common symptom and usually has to be elicited on interrogation. The light sense, particularly the perception of the light difference, is reduced. There is difficulty in seeing small red or green colored objects, and the vision is usually better when it is bilateral and is tested in a subdued light. I shall leave out of the discussion of retrobulbar optic neuritis types of purely exogenous toxic origin, such as those due to tobacco, alcohol, lead, carbon bisulfide, thallium acetate, fusel oil, etc., and likewise those due to endogenous poisons, such as are associated with diabetes, beriberi, cutaneous burns, internal hemor-

rhage and pregnancy. Also there can be excluded, I think, those cases associated with Leber's disease, vascular sclerosis (aneurysm), excessive ingestion of quinine and cranial polyneuritis. These etiologic factors are fairly well established or can be definitely determined without much difficulty.

I should like to confine myself, then, to a few remarks concerning multiple sclerosis, other demyelinating diseases and sinusitis or focal infections, of which sinusitis is apparently the most important, as factors in producing retrobulbar optic neuritis. I believe, furthermore, that one may be justified in formulating a hypothesis which will connect multiple sclerosis, sinusitis and chiasmic arachnoiditis.

The controversy which has raged for many years, ever since the publication in 1817 of Beer's article "On Vicarious Blindness from Suppressed Snuffles, Without Evident Accumulation of Mucus in the Frontal Sinus" to the discussion of Campbell's recent article on "Relationship of Sinusitis to Optic and Retrobulbar Optic Neuritis," has shown a swinging of the pendulum back and forth between the two conditions as etiologic factors for retrobulbar optic neuritis. Campbell's article bears careful attention by those who are interested in the subject. Recent studies by Frost indicate an important relation between disease of the posterior sinuses and papilledema. Other authors have reported acute blindness due to suppurative sinusitis. Onodi in 1908, Loeb in 1909 and Schaeffer in 1920 pointed out the close anatomic relation between the optic nerves and the posterior sinuses. The theory that the optic nerve is affected by direct extension through a thin pathologic bone from the sinus is probably not the entire story. Batson's recent anatomic studies appear to throw further light on the subject. In a review of the venous connections between the eye and the sinuses he stated that:

> . . . the many direct connections of the veins of the nose and paranasal sinuses with the veins of the orbit and cavernous sinuses of the cranium seem to present the probable pathway for the involvement of the orbit in infectious conditions of the nose.

Eagleton (1932) emphasized the close connection between otitis and infected vessels of the sphenoid sinus and the relation of otitis to meningitis as a result of showers of infected emboli. Campbell, in speaking of the status of the sinuses as a causative agent, stated:

> This condition appears still to be in the stage where individual opinions and theories prevail, and a great deal of real clinical and probably experimental research will be necessary to place the status of the sinuses in disturbances of the optic nerve on a foundation acceptable to all.

The question of the causation and pathogenesis of multiple sclerosis is as yet unsolved. Nowdays many neurologists think that all demye-

linating diseases (acute disseminated encephalomyelitis, acute ascending myelitis, neuromyelitis optica and diffuse periaxial encephalitis or Schilder's disease) are but varieties of multiple sclerosis. Certainly retrobulbar optic neuritis is a common, if not the most common and early, symptom in these diseases. Various authors have estimated that retrobulbar optic neuritis is the first manifestation of multiple sclerosis in from 11 to 33 per cent of cases. The European authors fix it at 60 per cent of cases.

A recent study by Tracy Putnam, of Boston, is, I think, of much value in arriving at the pathogenesis of multiple sclerosis. His experiments show that venous obstruction leads to axon degeneration and glial proliferation distributed in patches throughout the white matter of nerve tissue. These patches of focal disturbance always occur in encephalitis, in the acute stage of postvaccinal encephalitis and in rabies. He also produced venous thrombi by allergic reactions and suggested that allergy may be a factor in the disease. Curiously enough, H. H. Stark in 1921 advanced the theory that anaphylaxis accounts for the involvement of the nerve from a focus of infection. He expressed the belief that there may be a sensitization of the tissues of both the sinus and the orbit by bacterial proteins, an allergy being produced which results in a localized anaphylactic reaction each time the person comes in contact with a fresh infection of the same bacteria in the nose, teeth or tonsils or some other part of the body. Ruedemann has under observation 2 persons who are sensitive to certain foods, in whom optic neuritis develops on ingestion of the allergin. Bedell has mentioned a case of petechial retinal hemorrhage in an allergic person. Therefore, I agree with Campbell that "the allergic state as a possible etiologic basis in some of these cases should not be dismissed without further thought and investigation."

But to return to Putnam's work. After many studies and experiments, he concluded that the patches of demyelination and glial proliferation seen in cases of multiple sclerosis are due to venous thrombi, which he has seen not only in the white matter but in other tissues of the body as well. He does not venture to guess why the thrombi are formed but limits himself strictly to pathogenesis.

In several cases of optochiasmic arachnoiditis, purulent sphenoiditis has been found. In 1 case reported recently by Worms, pus had broken through the sphenoid sinus and involved the chiasm. This occurred five years after the attack of bilateral optic neuritis and blindness. Autopsy revealed the two optic nerves and the chiasm encircled with a mass of filamentary adhesions fixed to the base of the brain. This case is thus tied up with the cases of arachnoiditis. Operation was performed in 2 cases of what was considered optochiasmic arachnoiditis.

In 1 case (Cossa) the condition turned out to be encephalitis and in the other (Vail), multiple sclerosis. This last case deserves a more detailed report.

A white married woman of 27 was first seen on June 29, 1934, complaining of blurred vision, pain and tenderness of the left eye of two days' duration. Five years before a similar attack occurred in the right eye, which, in spite of an operation on the sphenoid sinus, at which nothing apparently was found, became entirely blind (no perception of light). Examination revealed atrophy of the optic nerve of the mixed type on the right and a normal disk on the left. The vision of the left eye was reduced to 20/40 and the visual field showed a small relative central scotoma. Since the possibility of early multiple sclerosis was considered, the patient was referred to a neurologist, Dr. H. D. McIntyre. According to his report there was weakness of the right side of the face. The remainder of the cranial nerves were normal. Deep reflexes were hyperactive on the two sides, those on the right being most pronounced. Abdominal reflexes were present but easily fatigued. A bilateral Hoffmann sign was present, which was more marked on the right. Ankle clonus was present on the right. Sensation was normal. Spinal puncture revealed normal pressure. The Wassermann reaction of the spinal fluid was negative, and the colloidal gold curve was normal. Analysis revealed a normal amount of globulin and 8 cells. The patient was given a course of typhoid vaccine intravenously, and four weeks later the vision in the left eye was 20/20 +, with recognition of colors faintly in the fixation point. Two weeks later she returned, stating that her vision had begun to fail again. It was found to be 20/50, and two days later she was able only to count fingers at 2 feet (60 cm.) in the nasal field. Craniotomy and exploration of the chiasmic area were performed by Dr. Nolan Carter. The right nerve was flat and yellowish, and the left was rounded, pink and edematous. An unusual amount of yellowish fluid was evacuated from the chiasmic cistern. No adhesions were found. Two weeks afterward, when the patient was discharged from the hospital, she could see light in the temporal field of the right eye (for the first time in five years) and was able to count fingers at 6 feet (182 cm.) eccentrically with the left, which presumably had a large absolute central scotoma. The nerve head was somewhat edematous and pale. She has been followed since the operation at various intervals, and there have developed diminished motor power on the two sides and somewhat spastic extremities, especially on the left. The abdominal reflexes are absent. Her vision has remained unchanged, and both disks show atrophy of the mixed type. The neurologic diagnosis was multiple sclerosis.

In the present state of knowledge treatment should be liberal. All foci of infections should be attacked, especially the posterior sinuses, and the allergic state and the use of foreign proteins and salicylates should be investigated. If no improvement occurs after attention to these factors, surgical exploration of the chiasmic region should be performed. The operative mortality is low as a rule, and the recovery or the improvement of vision (in more than 50 per cent of the cases reported) is frequently assured, especially when the operation is undertaken soon enough. In many cases, particularly when the disks are atrophic, the recovery of vision is likely to be slow, so that one must not despair if

the vision remains poor for several months. It is possible, however, to say that if no vision is recovered within six months after operation the prognosis is hopeless.

CONCLUSIONS

Optochiasmic arachnoiditis exists as a clinical entity, presenting certain signs and symptoms which on careful study may yield a preoperative diagnosis. At any rate, the signs point to some involvement of the chiasmic region and warrant surgical exploration, at which time the diagnosis can be made. Case reports indicate that surgical intervention is justified on the ground of improved vision in most of the cases.

The posterior nasal sinuses may be the portal of entry for the toxin or virus, and recent evidence indicates that encephalitis, multiple sclerosis and the associated demyelination diseases as well as optochiasmic arachnoiditis may all be closely allied and may be different manifestations of the same pathogenic process, which could well be thrombi in the venous system of the nerve tissue. The many cases of retrobulbar optic neuritis in which dramatic improvement of vision occurs after treatment of the sinuses, whether by surgical or by medical hyperemic procedures, justify these steps.

It is too soon to say that signs of multiple sclerosis will not develop later in cases of optochiasmic arachnoiditis.

BIBLIOGRAPHY

Arce, J.: Optochiasmatic Arachnoiditis (Syndrome of Balado), Bull. Acad. de méd., Buenos Aires **111**:106, 1934.

Balado, M., and Satanowsky, P.: Surgical Treatment of Atrophy of the Papilla, Arch. argent. de neurol. **4**:71, 1929.

Batson, O. V.: Relationship of the Eye to the Paranasal Sinuses, Arch. Ophth. **16**:322 (Aug.) 1936.

Bedell, A. J.: Stereoscopic Fundus Photography, Tr. Sect. Ophth., A. M. A., 1935, p. 17.

Beer, cited by White.

Bollack, J.; David, M., and Puech, P.: Optochiasmatic Arachnoiditis, Paris, Masson & Cie, 1937.

Campbell, E. H.: Relationship of Sinusitis to Optic and Retrobulbar Neuritis, Arch. Ophth. **16**:236 (Aug.) 1936.

Cossa, cited by François.

Craig, W. M., and Lillie, W. J.: Chiasmal Syndrome Produced by Chronic Local Arachnoiditis, Arch. Ophth. **5**:558 (April) 1931.

Cushing, W., and Eisenhardt, L.: Meningiomas Arising from the Tuberculum Sellae, Arch. Ophth. **1**:168 (Feb.) 1929.

Eagleton, W. P.: Suppurative Meningitis of Otitic and Nasal Origin, Arch. Otolaryng. **15**:885 (June) 1932.

Fralick, F. B.: Abstract from a course in retrobulbar optic neuritis given before the American Academy of Ophthalmology and Otolaryngology, 1936.

François, J.: Contribution to the Study of Optochiasmic Arachnoiditis, J. belge de neurol. et de psychiat. **35**:188, 1935.

Frost, A.: Papilledema, with Special Reference to Papilledema Associated with Sinus Disease, Tr. Am. Ophth. Soc. **33**:480, 1935.

Heuer, G. J., and Vail, D. T., Jr.: Chronic Cisternal Arachnoiditis Producing Symptoms of Involvement of the Optic Nerves and Chiasm, Arch. Ophth. **5**:334 (March) 1931.

Loeb, cited by Campbell.

Onodi, cited by Campbell.

Puech and Mahoudeau, cited by Bollack, David and Puech.

Putnam, T.: Etiologic Factors in Multiple Sclerosis, Ann. Int. Med. **9**:854, 1936.

Rollet, cited by François.

Ruedemann, A.: Personal communication to the author.

Schaeffer, cited by Campbell.

Stark, cited by Campbell.

Vincent, C.; David, M., and Puech, P.: In Regard to Seven Cases of Optochiasmatic Arachnoiditis, Rev. neurol. **1**:760, 1931.

White, L. E.: Loss of Sight from Posterior Accessory Sinus Disease, with Report of Three Cases, Boston M. & S. J. **176**:891, 1917.

Worms, G.: Oculo-Hypophyseal Syndrome Secondary to a Suppurated Sphenoidal Sinus, Arch. d'opht. **53**:207, 1936.

RESEARCH STUDIES OF THE EYE

SOME TECHNICAL AND PRACTICAL NOTES

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In this article I do not claim to report any innovations but simply to call attention to some guiding principles of technic which have aided me greatly in histologic research in the field of ophthalmology.

Since I am forced to work in a small private laboratory that lacks the substantial equipment of an endowed research institution and since I am able to devote to scientific research only such time as a professional practice permits, I have found it in some measure expedient to adapt my research technic to these conditions. Accordingly, these notes, although of no value to workers in large laboratories, may be of some use to men who, like myself, must carry on in a small laboratory.

FIXING AND PRESERVATION OF SPECIMENS

Although solutions of formaldehyde are generally recognized as among the most unsatisfactory of fixatives and the disadvantages of their use as preservative fluids for anatomic specimens are known to all, nevertheless these solutions are still widely used by ophthalmologists.

In 1925, while at Bologna, Italy, I had occasion to frequent the histologic laboratory of Professor Ruffini. After having examined the collection of histologic material which had been housed there for years, I asked the director's advice with respect to the most satisfactory methods of preserving ophthalmologic material for use in pathologico-anatomic as well as purely morphologic studies.

Professor Ruffini showed me certain embryonal specimens which had been preserved for more than twenty years and which, so he told me, could still be put to admirable use in detailed cytologic research. He also provided me with notes on his technic of fixing and preserving; these, although unpublished at the time, appeared in print some years later.

Ever since learning Professor Ruffini's technic, I have applied it in my own studies and in preserving my material. I find it superior to all others. First, in following meticulously the rules laid down by Professor Ruffini one obtains an excellent fixation which permits of the most varied staining and insures preservation of pathologicoanatomic specimens for years. Moreover, these specimens will retain the suppleness of fresh tissue and may be utilized for detailed histologic studies at any time.

The sole disadvantage of the method is that it requires a good deal of time, especially if large specimens are to be fixed. Consequently, it becomes impracticable when there is urgent need for an early pathologicoanatomic report as an aid in diagnosis.

The fluid which Professor Ruffini recommended as a general fixative is designated "R. 3" in his brochure. There are, as a matter of fact, two other liquids, R. 1 and R. 2, used, respectively, for fixing the eggs of batrachians and the larvae of anurous amphibians.[1]

The formula for Professor Ruffini's fluid is as follows:

	Gm. or Cc.
Müller's fluid (2 Gm. of potassium dichromate and 1 Gm. of sodium sulfate in 100 cc. of water)	50
Solution of chromic acid, 1 per cent	50
Glacial acetic acid	2
Mercury bichloride	1

The acetic acid can be added immediately, since it does not alter the fluid. On occasion, however, the fluid may turn brown and lose its efficacy. I attribute this phenomenon to the impurity of the reagents. If this discoloration does not take place within the first few days, there is no likelihood of its occurrence even at the end of several months.

This fixative is markedly hypertonic, and for this reason Professor Ruffini advised that in the preparation of delicate tissues the specimens should first be fixed for several hours in a dilute fixative and then placed in the fixative of full strength. As to ocular tissues, I have found it an excellent procedure to submit them first to a fixing fluid diluted to 50 per cent with distilled water. For small specimens of tissue, this preliminary process should last for from two to three hours. Whole eyeballs should remain in the weakened fixative for twenty-four hours; in fixing these organs it is well to make the injection into the vitreous by means of a platinum needle. The needle should penetrate along the equator in the superior or lateral segment. The site of perforation should be in the segment which will later be discarded, so that no mark will be perceptible in the cut sections. Enough fluid is injected —but the injection should not be made with too much force—to distend the eyeball that has collapsed after enucleation. After it has remained for from two to three hours in the diluted fluid, suspended by a thread

1. F. Caramazza (Rassegna ital. d'ottal. **2**:1299 [Dec.] 1933), in his unwarranted criticism of my studies of filmy sediment on the crystalline lens capsule and on the iris (Arch. f. Ophth. **119**:135, 1927), sought to display his familiarity with Ruffini's methods and stated that I referred to the fixative of Ruffini without specifying to which fluid I made allusion. Had he read Ruffini's brochure with any degree of attention he would have noted that only one formula is recommended for preservation of human material.

tied to a tendon as a precaution against deformation, the eyeball will have become sufficiently firm to permit the removal of a segment from the walls in order to facilitate penetration of the fluid. An ordinary razor blade serves admirably for the removal of this segment. Subsequently, the eyeball is left suspended in the fluid proper for twenty-four hours.

After fixation in the diluted fluid, all specimens, large and small, are placed in the undiluted fluid for twenty-four hours. In the preparation of eyeballs it is well to remove a second segment opposite the first; this facilitates future treatment.

Coagulation without shrinkage of the vitreous is a phenomenon commonly observed after application of the fixative; this always occurs if the vitreous has been invaded by exudate.

The fixing process is followed by a lavage of twenty-four hours' duration under running water. After this, the specimens are regularly treated with Kaiserling solution II, the formula of which follows:

	Gm. or Cc.
Potassium acetate	50
Glycerin	100
Alcohol, 95 per cent solution	100
Distilled water	500

In this fluid the preparations not only resume their original suppleness but are rid of the chromic salts. As Professor Ruffini remarked, this loss is not the result of simple diffusion but of a veritable process of replacement similar to that which is observed in living tissue when, for example, ions of potassium are substituted for the ions of sodium. Here the chromium is replaced by potassium, a substitution which, if it is thoroughly carried out, makes it easier for one to attempt histologic staining which cannot be obtained on material fixed in fluid containing chromic salts.

Kaiserling solution, too, is strongly hypertonic. Professor Ruffini advised treating the preparations in a series of solutions of graduated concentration if one is to avoid too rapid changes in osmotic tension. He recommended placing specimens in each of the following dilutions for twenty-four hours: 1 part Kaiserling solution to 3 parts distilled water; 1 part Kaiserling solution to 1 part water; 2 parts Kaiserling solution to 1 part water, and undiluted Kaiserling solution. In order to eliminate the mercury bichloride, there should be added to the foregoing solutions a few drops of a concentrated solution of potassium iodide (10 Gm. of potassium iodide to 10 Gm. of water).

As I stated previously, tissues placed in Kaiserling solutions will be rid of the chromic salts. If the pieces of tissue are small, the fourth dilution will already be colorless. But in fixing whole eyeballs, par-

ticularly if the vitreous body, filled with exudate, has assumed a certain firmness, it is frequently necessary to change the fourth concentration several times before the fluid within the tissue will remain colorless for twenty-four hours, that is to say, before one has obtained complete extraction of the chromic salts.

This step tends to prolong the fixing procedure and may on occasion present a serious handicap in addition to entailing a great deal of expense in those countries in which alcohol and glycerin command high prices.

After the specimens are free from the chromic salts, they are allowed to remain in the Kaiserling solution. In this manner suppleness is preserved for many years with no modification of the histologic structure or tinctorial properties of the various tissues.

For the process of rendering the specimens transparent after dehydration, I prefer to use benzene because of its great volatility. Professor Ruffini is opposed to the use of xylene and benzene; he expressed the belief that these substances render the tissues hard and friable. He recommended, however, that after the use of various solutions of alcohol a mixture of equal parts of cedar-wood oil and oil of bergamot be employed. I have never used this mixture for eyeballs for I personally prefer embedding in pyroxylin. On the other hand, I have placed smaller specimens of tissue in the mixture and readily observed its advantages, for with its aid I have been able to cut specimens of the human cornea into serial sections of from 6 to 8 microns.

As I have already mentioned, one of the principal advantages of the technic just described consists of its adaptability to the most diverse methods of staining. For example, after following the outlined procedure one may obtain excellent stainings with the methods of Giemsa and of Bielschowsky.

For ordinary staining with hematoxylin and eosin, I use Carazzi's hematoxylin, a staining solution that resembles the hemalum of Meyer. It offers two fundamental advantages: (*a*) It matures rapidly on account of its potassium iodate content (and this permits it to be utilized only a few hours after it has been made up). But in certain climates, that of São Paulo, for example, it is necessary to refrain from adding this salt, as it causes it to age rapidly and become useless. Without this salt, on the contrary, it matures in from three to four days and preserves its tinctorial properties for several months. (*b*) It never overstains the preparations, and no differentiation is necessary—two factors of definite importance to the worker who does not always have abundant time at his disposal. The sections remain in the staining solution for from ten to twenty minutes or more and are then washed in running water or placed in a large evaporating dish that contains tap water.

The formula of Carazzi's hematoxylin is as follows:

	Gm. or Cc.
Powdered hematoxylin	0.50
Potassium iodicum (KIO_3)	0.01
Potassium alum	25.00
Glycerin	100.00
Distilled water	500.00

Dissolve at room temperature and stir frequently.

For staining with eosin, I use a 1 per cent aqueous solution in which I allow the sections to stand for from twenty-four to forty-eight hours. I then wash the tissues in distilled water and differentiate them in alcohol (rapid passage through 70 per cent alcohol and 95 per cent alcohol and twice through absolute alcohol). The preparation loses the excessive coloration without danger of complete decoloration (such as occurs in too rapid stainings). I obtain, in addition, a clear differentiation of the various protoplasmic and nucleolar parts (fig. 1).

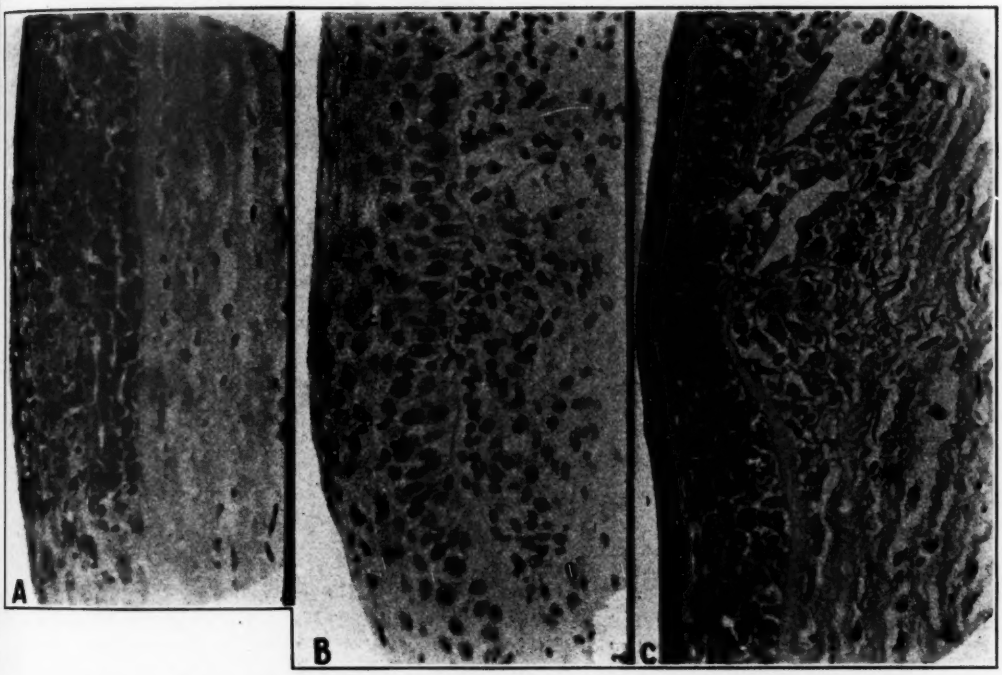

Fig. 1.—Corneal tissues fixed in Ruffini's fluid and stained with hematoxylin and eosin. *A*, trachomatous pustule of the cornea. The normal and altered epithelial cells and polymorphonuclears that have immigrated into the epithelium are well differentiated by the staining. *B*, extremity of a trachomatous corneal pannus. The structure of the nuclei is plainly distinguishable. *C*, extremity of a trachomatous corneal pannus. Edema of the epithelium and the different types of cells which go to form the extremity of the pannus and the vascular wall are plainly visible.

If the tissues have been fixed according to the methods described, silver impregnation by Bielschowsky's method (fig. 2) will provide results inferior to those obtained after fixation in Flemming's solution. I have found that impregnation is easier and more thorough after double embedding in pyroxylin and in paraffin. However, in order to obtain satisfactory silver staining it is often necessary to keep the sections in a solution of silver hydrate for a long period. I employ Bielschowsky's staining fluid (or better still Achucarro's formula) in undiluted form and

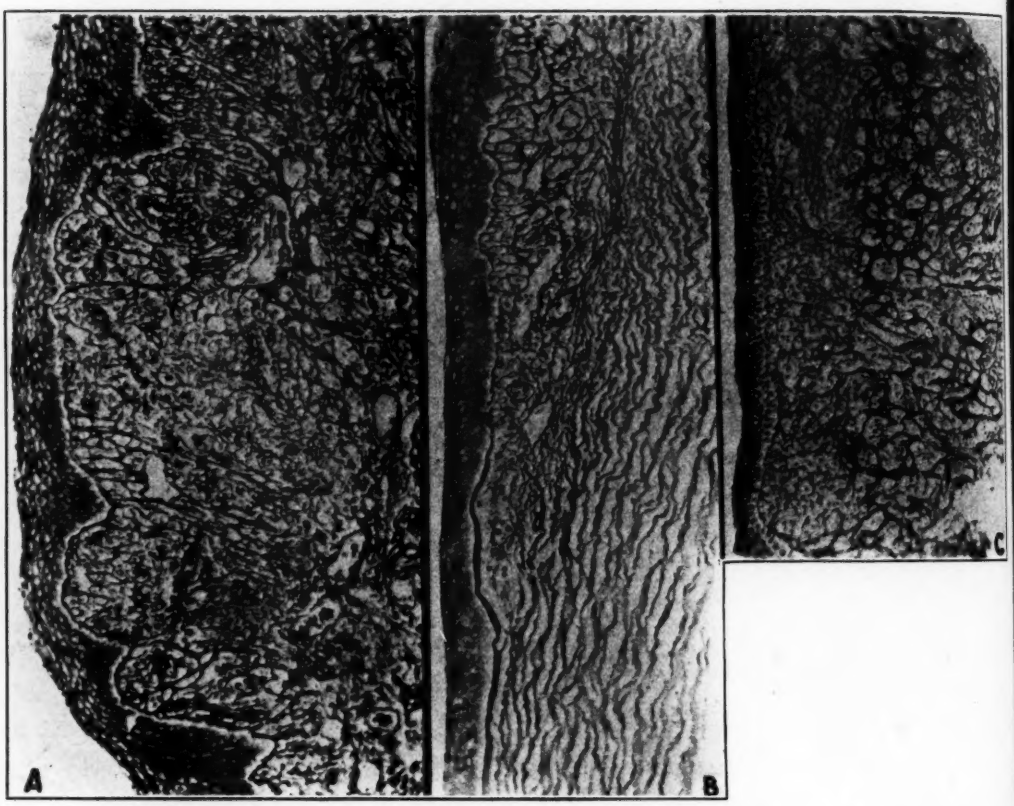

Fig. 2.—Corneal tissues fixed in Ruffini's fluid and stained by Bielschowsky's method: *A*, trachomatous tissue from the limbic region; connective and reticular tissue are present. *B*, extremity of a trachomatous corneal pannus; modifications of the corneal lamellae and of the lamina elastica anterior. *C*, trachomatous corneal pannus; modification of the infiltrated corneal lamellae.

leave the sections in it for one hour. I find it necessary now and then to double or even to treble the amount of time allowed for silver staining.

It is well in each instance to check the time required for impregnation by means of a small slide prepared expressly for this purpose; one

should keep in mind that granular reaction in the fibrils indicates that the time allotted has been inadequate. If the time required for impregnation is once determined for a certain type of tissue in a certain type of solution, the results will be absolutely constant.

Whenever I wish to examine the same tissue specimen under different stains, I make serial sections and then prepare four or more series in the following manner: I have before me four slides, on each of which I pour a small amount of distilled water and place the section obtained by the microtome directly on them. On the first slide I place the sections corresponding to the numbers 1, 5, 9, 13 and so on; on the second slide, sections numbered 2, 6, 10, 14 and so on; on the third

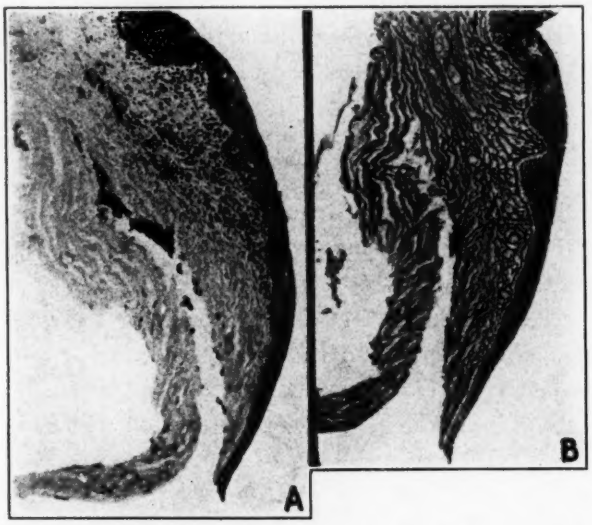

Fig. 3.—Trachoma of the lunula and an initial trachomatous nodule. The two sections, cut a few microns apart, were stained, respectively, with hematoxylin and eosin (*A*) and by means of silver impregnation (*B*).

slide, sections numbered 3, 7, 11, 15 and so on, and on the fourth slide sections numbered 4, 8, 12, 16 and so on. When the slides are full, I add several drops of water and place the slides in the incubator, where they remain for a number of minutes at 52 C., a temperature calculated to distend the sections both satisfactorily and quickly. I leave them in the incubator at 37 C. for from twenty-four to forty-eight hours. This distribution of the series enables me to examine sections stained by four different processes, which are separated from one another by only a few microns. Thus, for all practical purposes it may be said that the identical section has been examined by different methods (fig. 3). The advantages of such a technic are manifest.

REMOVAL OF CORNEAL TISSUE FOR BIOPSY

The removal of tissue for pathologic study is seldom applied to the cornea, no doubt because of the common feeling that it is dangerous to touch the cornea. Yet from my own experience I have found that nothing is more innocuous, even for the cornea itself, than skilful removal of tissue. Needless to say, one should not think of attempting to remove tissue from the pupillary field, as the remaining corneal facet would cause functional impairment.

In addition to the induction of complete anesthesia before the removal of tissue, there are two precautionary considerations: preliminary disinfection of the conjunctival sac and the use of well sharpened knives.

I am accustomed to begin disinfection of the conjunctival sac two or three days prior to removal of tissue. In the morning the parts are painted with a 2 per cent solution of silver nitrate; about ten minutes later the conjunctival sac is washed, the eyelids being inverted. When this is done, I introduce an ointment containing Besredka's antivirus into the conjunctival sac, and in the evening I instil a 4 per cent solution of mercurochrome. Before removing any tissue I wash the conjunctival sac with a substantial quantity of physiologic solution of sodium chloride. With these precautions I have never encountered infections in the course of removal of more than 100 biopsy specimens, the majority of which were taken from trachomatous eyes.

For a cutting instrument, I use an iridectomy knife; it must be well sharpened, and I never use the same knife for removal of more than one or two specimens of tissue. The eyeball is immobilized by grasping it with a fixation forceps such as is used in cataract operations; this is done in a sector distal from that in which the tissue is to be removed. Tissue is removed from the central zone of the cornea toward the limbic zone. The tip of the lancet is inserted almost perpendicularly at the extremity of the zone to be removed, and when it has penetrated the superficial tissue layers it is turned so that its surface becomes virtually parallel to that of the cornea; with the lancet in this position, one advances directly toward the limbic region, forcing oneself to follow the curvature of the cornea. When the point of the instrument has penetrated beyond the limbic region, one of the borders of the piece to be removed is freed by a lateral movement; the lancet again penetrates beneath the piece of tissue and frees the other border by a second lateral movement. If the tissue still adheres to the side of the conjunctiva, it may be detached by means of iris forceps and scissors. In this manner sizeable specimens can be procured (fig. 4).

If the cornea is well infiltrated, removal of tissue is extremely easy, as the tissue offers no resistance. Conversely, in a cornea that is only slightly infiltrated one often encounters formidable resistance; hence the need of an excellent cutting instrument.

It goes without saying that the procedure described can be done only in approximately the anterior half of the cornea, since perforation would be impossible, and too deep an incision may produce ectasia. It is well before undertaking removal of tissue from the cornea to study the corneal thickness by means of the slit lamp. If the cornea has

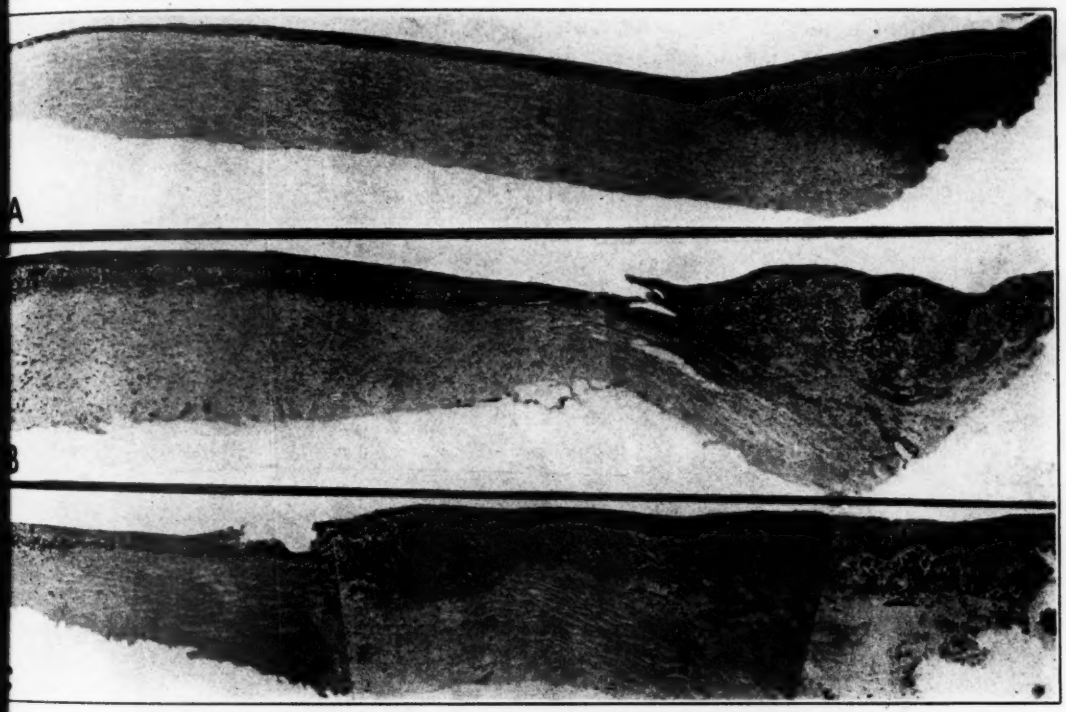

Fig. 4—Biopsy specimen of the cornea showing: *A*, an initial trachomatous corneal pannus and avascular trachomatous keratitis; *B*, a well developed trachomatous corneal pannus with papillary formation in the limbic region and discrete parenchymatous infiltration; *C*, trachoma of the lunula, a nodule of the limbic region and corneal pannus.

already been rendered extremely thin by pathologic processes, removal of tissue will be difficult or even impossible.

I have removed corneal tissue for biopsy numerous times, and only twice has the tip of the lancet accidentally penetrated the anterior chamber, and then the corneas were thin. In both instances, as soon as the anterior chamber began to empty I withdrew the instrument without removing any tissue, and the incident was inconsequential.

When I have completed the removal of tissue, I instil a solution of mercurochrome into the conjunctival sac and introduce an ointment containing an antivirus; then I apply a dressing, which is renewed daily for three or four days. This length of time is sufficient for the cornea to be covered by epithelium, even if the loss of substance is considerable.

In connection with the removal of tissue for biopsy, a small false pterygium may be formed. Sometimes a corneal facet remains which is often more transparent than the adjacent zones but which, being situated at the periphery, is of no significance to corneal function.

PLACEMENT OF LANDMARKS ON CONJUNCTIVAL OR CORNEAL TISSUE

Whoever has devoted himself to the comparative study of the biomicroscopic and histologic aspects of tissue knows how difficult it may be to find again minute lesions, even on serial histologic sections, especially if the pathologicoanatomic character of such lesions is unknown.

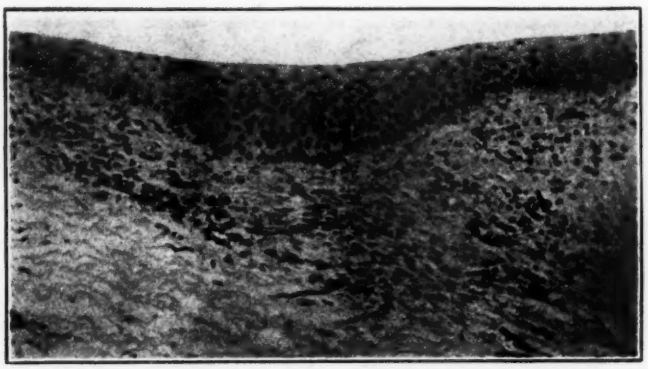

Fig. 5.—Section of corneal tissue demonstrating the value of tattooing. An abundance of india ink remains in the epithelial cells along the path of the needle (center of the fossette) and in the cells of the subjacent connective tissue.

I encountered this difficulty when I first attempted to study the parenchymatous vessels, which project forward into the cornea, and the structure of Herbert's extremity of pits. It then occurred to me to employ tattoo marks of india ink, which would serve as landmarks and which I could execute under the slit lamp. Although it would be superfluous to describe the technic of tattooing, which is so well known, I should like to outline certain technical points applicable to specific objectives.

In cases in which enucleation of the eyeball is urgent, tattooing can be done the night before the operation or even at the start of the operation. It is a useful procedure to mark two points, one on either side of the area of special interest. These points will be so situated that the line which unites them will indicate the precise course to follow in

sectioning the tissue with the microtome. A single prick with the tatooing needle suffices to leave behind enough india ink to identify in the specimens and the histologic sections the particular segments of tissue in which one may be interested.

If it is possible or advantageous to allow an interval between the time of tattooing and that of removal of the tissue (as is often the case when one has to deal with a small biopsy specimen), it is preferable to make three or four punctures with the tattooing needle, for, as every one knows, some of the ink will be lost during the first days. Figure 5 shows tissue which was tattooed under these conditions.

Tattooing can likewise be profitably employed for fixed specimens, as in the small lesion produced by the needle there remains a quantity of ink sufficient to serve as a guide when the piece is sectioned by the microtome. Furthermore, in histologic sections one sees only a few granules of ink running free in the edges of the slight solution of continuity. The method is serviceable, since often slight solutions of continuity suffice to put one on the trail of the points in which one is interested.

ADVANTAGES OF USE OF COAGULANTS IN OCULAR OPERATIONS

ESPECIALLY IN EXTRACTION OF CATARACT AND IN PLASTIC OPERATIONS

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For several years I have been accustomed to use coagulants in surgical operations on the eyeball and its adnexa. The excellent results that I obtain with such agents and the fact that they are so little utilized, at least so far as I have been able to ascertain, prompted me to call the attention of my colleagues to a technic which, without warrant, has been generally neglected.

The objection may be raised that skilful and opportune administration of epinephrine hydrochloride or the use of thermocautery suffices to prevent or to inhibit the minute hemorrhages which may complicate an otherwise adroitly and expeditiously performed operation. These two measures represent completely disparate mechanisms.

Epinephrine, as every one knows, inhibits hemorrhage by the contractile effect exerted on the vascular walls and the concomitant constriction of the lumen. But the drug has no influence on atheromatous vascular walls, and this explains why the hemostatic effect appears so slight when the drug is administered to aged persons who are markedly arteriosclerotic. Moreover, as is well known, vasoconstriction is succeeded by a further effect, vasodilatation, which gives rise to secondary hemorrhages. Still one must not forget that this state of vasodilatation is anticipated and exacerbated by the administration of atropine and that this circumstance renders dangerous the instillation of the medicament directly after an operation in which epinephrine has been used.

Cauterization inhibits hemorrhage by the production of an eschar, which serves to occlude the vascular lumens, or by coalescence of the walls, the effect of a coaction. Yet there is no denying that this procedure, to which Denig [1] referred favorably in a recent article, is not always practicable, for example, if hemorrhage of iritic origin is present. Besides, even if cauterization is done with a loop heated at the lamp, the eschar produced, although extremely slight, still represents a source of inflammation which may retard the process of cicatrization.

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1. Denig, R.: Die Iridotorsion im Vergleiche mit der Trepanation der Lagraneschen Operation und der Zyklodialyse, Klin. Monatsbl. f. Augenh. **99**:1 (July) 1937.

The action of a coagulant is altogether different from that of cauterization. With the use of such an agent a sufficiently tenacious coagulum rapidly forms, which occludes the severed vessels. In this way one merely accelerates and reenforces the physiologic mechanism which tends to arrest hemorrhage.

I have long used a proprietary coagulant from blood platelets (coagulen) for this purpose. Motivated by the usual feeling of fidelity to the methods which lead to favorable results, I have refrained from experimentation with other products, but it is likely that the same results would be achieved by means of similar substances or with the use of purified snake venom, which has recently been introduced into surgical practice as a hemostatic.

For operations on the eyeball, I prescribe the injection of 20 cc. of the coagulant into the subcutaneous tissue of the abdominal wall. Experimentation has shown me that a satisfactory effect is attained within two hours. It always seems to me that the action is more pronounced after a long interval. Accordingly, if I am to operate early in the morning I regularly administer the coagulant the evening before, but if an operation is scheduled for late in the day the patient receives the coagulant during the early hours of the morning.

For all operations in which the eyeball is not excised, I prefer the local application of the coagulant in its powdered form. At the proper moment a bit of powder is spread over the operative field. I then wait for the formation of a dense coagulum, which is removed before resumption of the surgical work.

It is not my intention to introduce any case histories, and I shall limit myself to a discussion of certain considerations and observations elicited by several years of practical experience.

Coagulants are useful in operations for the extraction of cataract, whether by the intracapsular or the extracapsular method.

As every ophthalmologist knows, when a conjunctival flap is made during keratotomy a considerable amount of blood will often penetrate the anterior chamber; in patients whose blood vessels are atheromatous and whose eyes are hypotonic, the surgeon may readily observe a small stream of extravasated blood penetrate and fill the anterior chamber; it may even happen that after he has succeeded in removing this initial extravasation of blood, a second, smaller stream will gather. Such incidents may be particularly annoying during operations on the iris. Hemorrhage sometimes continues from the iritic vessels even after the operation is completed; this is particularly likely to occur in cases of complicated cataract. But although hemorrhage of this sort may mar the smooth course of an iridectomy or make difficult the grasping of the capsule of the lens during an intracapsular extraction of cataract, it is most likely to cause serious consequences during extracapsular extraction.

In fact, whereas the blood deposited on the anterior surface of the iris is, as a general rule, absorbed with great ease, that which remains on the capsular surface is slow to disappear, and if it forms into masses, complications always arise. If the latter occurs, one observes over a period of several days minute coagula between the masses or on their surfaces. It is noted that the masses, which probably will be covered with a fibrinous sediment, are reabsorbed with difficulty. In general, if the coagulum is slightly larger, absorption no longer takes place, but instead the organization of the coagulum itself forms a synechia between the periphery of the iris and the remnants of capsular and crystalline substance. This marks the beginning of a secondary cataract.

But, if one has administered a preliminary injection of a coagulant and if after keratotomy one waits a minute or two, it will be observed that an extremely dense coagulum will form, which may have to be removed with the forceps. Blood that has extravasated into the anterior chamber or which appears during an operation on the iris will also coagulate rapidly. Usually this coagulum can be removed by lavage of the anterior chamber; this is done by the introduction of a Daviel spoon and by directing to the part of the spoon remaining outside of the chamber a fine jet of physiologic solution of sodium chloride. More rarely, one has to extract the coagulum by means of the forceps. After the coagulum has been removed, no more blood is observed to extravasate into the anterior chamber; hemostasis remains complete.

The tenacity of the coagulum renders more solid the adhesion of the conjunctival flap and facilitates the formation of the anterior chamber. Another slight advantage to be obtained with the use of coagulants is that if one employs the Elschnig fixation forceps to grasp the episclera, the hemorrhage that is readily induced by this instrument in the region of fixation will be immediately arrested.

The use of coagulants offers the same advantages in operations for glaucoma as were mentioned in connection with the conjunctival flap employed in cataract operations; however, during the operation proper, the advantages are less important if one is accustomed to work under a fine jet of physiologic solution of sodium chloride, which will constantly keep the operative field free from blood.

After enucleation, a small amount of coagulant introduced into the orbital cavity, followed by compression of several minutes' duration through the eyelids and, finally, the application of a compressive dressing, obviates completely the extravasation of blood which is so often present, particularly in elderly patients, and which frequently involves the loose connective tissue of the other eye.

In various operations in the adnexa (such as an operation on the eyelids and the lacrimal sac or an operation for pterygium) the timely administration of a bit of powdered coagulant permits one to work in a

perfectly drained field and obviates the danger of minute secondary hemorrhages, which are often so troublesome.

But the field in which the use of coagulants renders the most remarkable services is that of plastic operation. I was first moved to experiment along this line following an extremely untoward incident that complicated a technically successful plastic operation. In view of the good results that I was able to obtain with the use of a coagulant, I decided to apply such an agent regularly.

I extirpated the nasal half of the lower eyelid of a man about 75 years of age, and then reconstructed the lid by rotation of the pedicle flap transplanted from the cheek. In order to combat a particularly irksome hemorrhage, I also resorted to the instillation of epinephrine hydrochloride in the operative field. About two hours after completion of the operation the bandage was completely soaked with blood, and soon blood began to drip from it. Efforts to check the bleeding by injections of hemostatics failed completely, and it was necessary to remove the dressing. A huge hematoma was found under the flap. Digital compression brought about cessation of the hemorrhage. However, as one might have expected, the flap became necrotic, and it was only by further surgical intervention that the deleterious sequels of this accident were in some small measure mitigated.

This was the instance which, as I already mentioned, decided me to use a coagulant, and local applications of the latter from the first have proved extremely effective. However, in a case in which I had administered a preliminary injection of coagulant, necrosis of the flap took place. After meditating over the contrast between this bad result and the truly excellent results obtained with local applications, I arrived at the following (perhaps erroneous) interpretation: Manipulations of the cutaneous flap (compression with the fingers or forceps; stretching the tissue) tend to produce traumatic vascular lesions; the coagulum that forms within such lesions will be extremely dense owing to the action of the (previously injected) coagulant and accordingly absorption will be difficult and the restoration of circulation throughout the flap will be retarded. After this one unfortunate incident, I abandoned the injection of a coagulant in plastic operations. I am therefore unable to state with certainty whether the poor results were a coincidence or whether such results would be the rule.

One more advantage assured by the use of coagulants in plastic operations is that the resultant solidity of the coagulum promotes coalescence of the flap with the underlying tissue, and this doubtless facilitates the "taking" of the graft.

The foregoing observations clearly demonstrate the great importance of the use of coagulants in ocular operations.

ROTATION OF THE CHEEK IN OPHTHALMOLOGY

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The method of rotation of the cheek consists in loosening the still healthy portion of the cheek, combined with more or less skin of the neck, for the purpose of restoring the contour of the face after all sorts of mutilations. These may be either small or large and may comprise mutilations of the upper and the lower lip, the nose, the cheeks and the lower eyelids. Even complete noses can be reconstructed. In such cases the inner cover of the nose can be prepared beforehand by an "epithelial inlay" placed under the skin of the cheek which is used to form the nose.

This loosening of the skin is done by making a large incision around the cheek, the natural lines of the face always being followed, i. e., the lines between the nose and the cheek and the cheek and the edge of the eyelid. The incision continues as far as the temple and descends along the ear at the side of the neck.

In this way the cheek, more or less complete, is utilized to restore the face, and the skin of the neck, which it drags with it, partly covers the cheek, forming a large wound in the neck instead of the original one which was in the face. This wound in the neck can always be closed by sutures, owing to the great elasticity of the skin of the neck, and in cases of extensive repair the head is inclined toward the shoulder and held there for several weeks, until the wound is completely healed.

I introduced this method in 1915. It is a general method and can be applied to all sorts of mutilations of the face. For this reason it is of great practical value even for the ordinary surgeon who has not specialized in plastic surgery, for the stomatologist and for the ophthalmologist.

As the cheek is rich in vessels, necrosis is almost impossible, even though there is tension in the portion of the cheek which has to cover the gap. If the rotation is well applied, no tension will exist in the part covering the gap, but the tension of the sutures round the neck will increase as they approach closer to the neck. Numerous sutures must join the turned cheek to the surrounding skin. The result is that the great tension which is caused by the strong rotation is divided among such a great number of sutures that there is only a small amount of tension on each one. For that reason the stitches must be made with the finest needle and the thinnest silk that can be found. This is why the scars scarcely show. The alternate sutures are taken away at the end of a week, and those that are left remain as long as is necessary, according to the tension, and are taken away gradually.

The general scheme varies with the size and shape of the defect. Figure 1 *A* shows the technic used in the reconstruction of the lower eyelid, and figure 1 *B* that used in the restoration of the upper eyelid.

REPORT OF CASES

CASE 1.—In 1937 I was called in by Professor Kondoleon, professor of surgery at the University Clinic in Athens, to see a girl 13 years old (fig. 2 *A* and *B*), who had burnt her face when younger. The left cheek had one long scar running along the nasolabial fold, but the right cheek was almost completely destroyed,

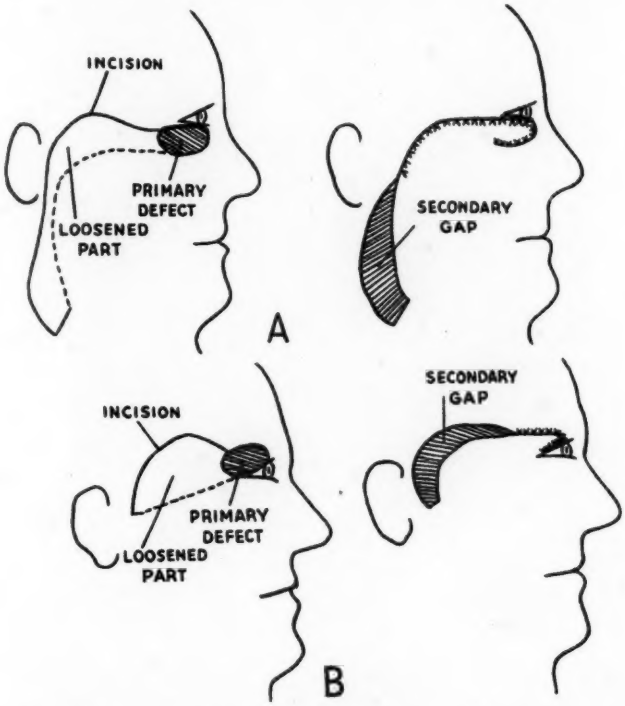

Fig. 1.—*A*, technic employed in the reconstruction of the lower eyelid; *B*, that employed in the reconstruction of the upper eyelid.

there being one large scar. She had a large ectropion of the lower eyelid and of the upper lip. I decided to apply my method of rotation of the cheek to repair both ectropions simultaneously and also to reconstruct the cheek with normal skin. The portions of the conjunctiva and the mucous membrane of the lip which were dragged toward the center of the cheek were loosened by incisions from the underlying scarred muscle tissue until they were completely free. The edge of the eyelid was then shortened to its normal length by the excision of a portion of its outer edge. This was necessary because the scars of the cheek had gradually lengthened the edge.

Great care had to be exerted to take away the scarred muscle tissue with the conjunctiva, because this might cause a repetition of the ectropions. The rotation had to provide the muscles and skin of the new lower eyelid, just as it had to

furnish new muscle tissue and skin for the lip, after the mucous membrane of the lip had been freed in the same way as the conjunctiva.

Figure 3 *A* shows the large defect in the cheek resulting from the operation and from the removal of all the scarred skin and muscle tissue from the right cheek. The dotted line in the sketch indicates the incision of the parts of the face

Fig. 2 (case 1).—*A* and *B*, patient before operation; *C* and *D*, patient some weeks after rotation.

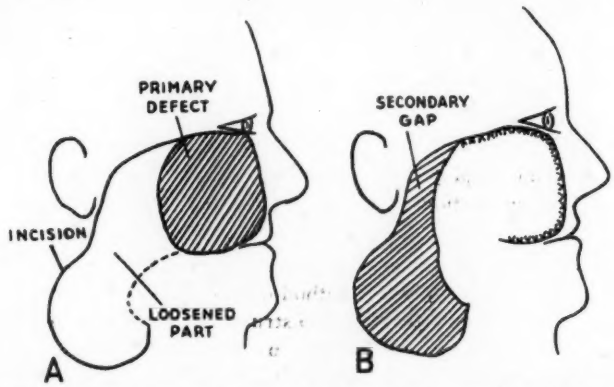

Fig. 3.—*A*, the first step of the operative technic employed in case 1; *B*, the second step.

and neck to be rotated, when they had been sufficiently loosened and made mobile enough to cover the large defect in the cheek. This rotation caused a secondary gap in the neck, which could be sewn together. Figure 3 *B* shows the primary gap being closed and the resulting secondary gap which had to be sewn together.

Fig. 4 (case 2).—*A*, photograph showing the inability of the patient to close his left eyelids; *B*, photograph showing method of rotation used, and *C*, photograph showing complete restoration of both eyelids.

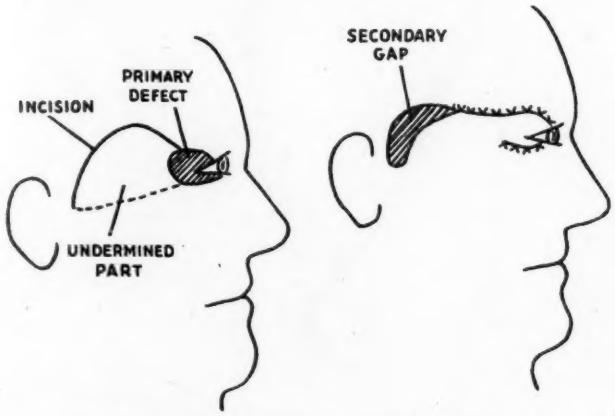

Fig. 5.—Operative technic employed in case 2.

Figure 2 *C* and *D* show the girl some weeks after the rotation without any corrective operation. The ectropion of the eyelid and that of the lip were radically healed; scars could scarcely be seen on the right cheek. The expression of the face was almost normal except for the curve of the mouth, which was caused by the new function of the muscle tissue brought from some distance away. Gradually the expression of the face will become normal in the course of a few months when these muscles have adapted themselves to their new functions.

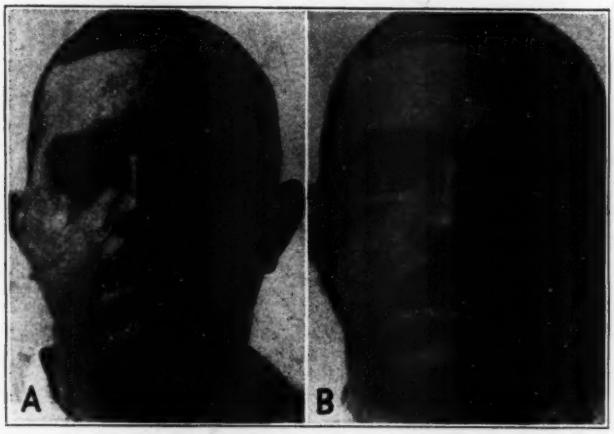

Fig. 6 (case 3).—*A*, patient before operation; *B*, patient after operation.

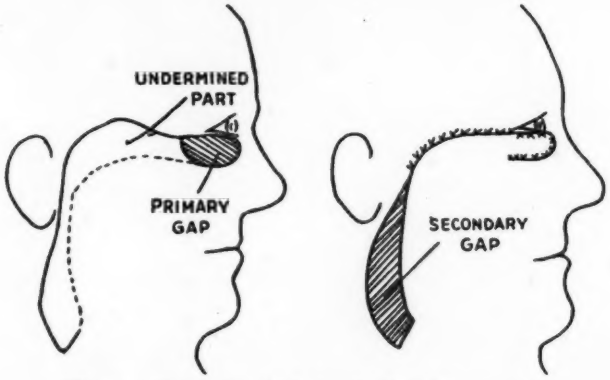

Fig. 7.—Operative technic employed in case 3.

Case 2.—Patient W had both eyelids of his left eye destroyed. In figure 4 *A* the patient is shown trying to close his eyes, but as the greater part of the left eyelids was missing he could not make them meet more than is pictured. Rotation of the cheek repaired both eyelids. This is the highest point to which rotation of the cheek can attain. The method used is clearly seen in figure 4 *B* and in figure 5. Figure 4 *C* shows both eyelids completely restored. Slight corrections from the esthetic point of view are still needed.

Case 3.—Patient E needed an entire right lower lid, as his had been completely destroyed (fig. 6 *A*). Figure 6 *B* and figure 7 show how rotation of the cheek was utilized for this purpose.

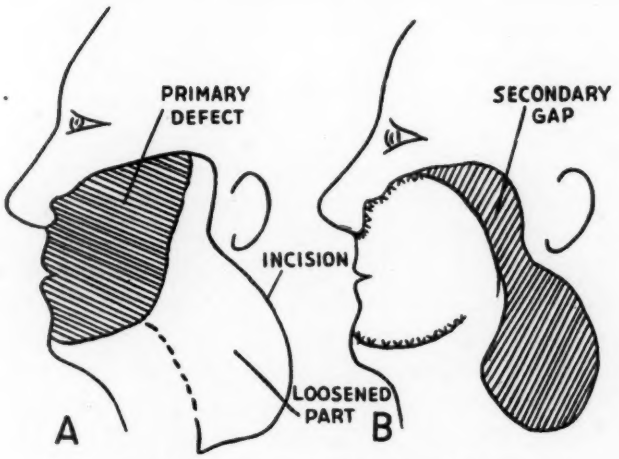

Fig. 8.—Operative technic employed in case 4.

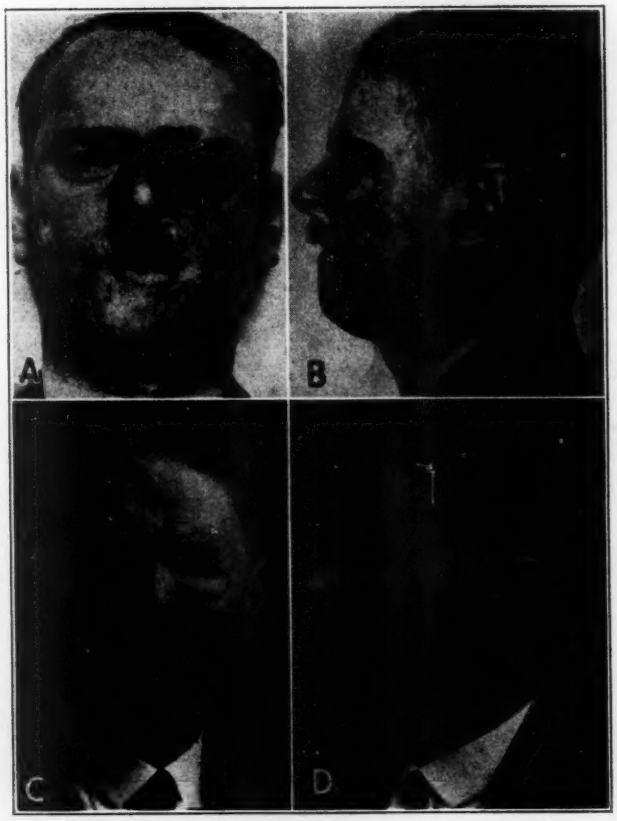

Fig. 9 (case 4).—*A* and *B*, patient before operation; *C* and *D*, patient after operation.

As the lower part of the socket was missing, an "epithelial inlay" was made to cover the inner side of the rotation flap in order to form the inner skin cover for the lower eyelid, so as to make it ready later to receive the artificial eye. In figure 6 *B* the artificial eye is already in place.

This "epithelial inlay," mentioned in the previous case, is a method I introduced over twenty years ago and is extensively used for plastic and structural surgical repair. It is also extremely valuable in the field of ophthalmology. The method consists in taking an exact model with stent (a material used by dentists) of wounds for which free skin grafts are to be used. When the stent becomes stiff, it is wrapped in a thin Thiersch graft, with the epidermis toward the mold. The mold then is replaced on the wound in exactly the same position as it was when taken and kept there under firm pressure for a week. This pressure prevents any secretion between the wound and the graft and in this way insures complete healing of the graft. With other methods these secretions loosen partly or completely the already attached graft. The thin grafts are taken from the inner side of the thigh; the resulting wound heals in a few days under a dry dressing.

I shall describe a case in which the gap is of the type that is the most difficult to repair; not that it is the largest, but it is so close to the neck that scarcely any part of the cheek is available and almost the entire rotation has to be made from the neck.

Case 4.—The patient, a French physician, was under the care of the well known ophthalmologist Morax, of Paris. The doctor was epileptic and had fallen into the fire, the left side of his face being entirely burned away. The eyelids were destroyed, and the cheek was replaced by a star-shaped scar. Morax had restored the eyelids, utilizing my method of biologic frontal flaps, i. e., pedicled flaps from the skin of the forehead, the pedicle not containing any skin but only veins, lymph vessels and nerves, all surrounding the frontal artery. These vessels remain protected by a layer of connective tissue. The advantage of this method is that the vessels of the elastic pedicle are never shut, even if the flap is turned at an angle of more than 180 degrees. Another advantage is that it is economical, as no skin tissue is wasted in the pedicle, the flap is mobile and there is no danger of necrosis.

After this operation Morax consulted me for the restoration of the left portion of the nose, the completely scarred cheek and the lower lip. I cut away the part shown in figure 8 and made an incision in the face and neck, as indicated in figure 8 *B*. The part surrounded by the cut was loosened and brought into the missing part of the face. The secondary gap was sewn together under great tension, and the head had to be inclined toward the shoulder for a week. Figure 9 *A* and *B* show the patient before operation, and figure 9 *C* and *D*, after operation. After such a large rotation of the cheek, corrective operation is usually necessary, but in this exceptional case the result was so satisfactory that the patient did not wish any further correction and is still delighted with the result.

BIOCHEMISTRY OF THE LENS

XII. STUDIES ON GLUTATHIONE IN THE CRYSTALLINE LENS

LAWRENCE ROSNER

C. J. FARMER

AND

JOHN BELLOWS

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The possible functions of glutathione in the lens as well as in the rest of the body have been a matter of discussion since the discovery of this substance by Hopkins.[1] Although much experimental work has been done on the glutathione in the lens, its actual isolation from this organ was only recently accomplished by two of us.[2] That glutathione has an important function in the metabolism of the lens appears likely from the work of Adams,[3] who showed that the oxygen uptake of a lens extract became nil when glutathione was dialyzed out; readdition of glutathione to the extract caused a resumption of its oxygen uptake. It was indicated that this substance is a constituent of an auto-oxidation system in which the sulfhydryl can act as a hydrogen donor, being oxidized to the disulfide form, which in turn can accept hydrogen from a "thermostable residue." It was shown by Goldschmidt [4] and Adams [3] that the beta cystallin of the lens is capable of acting as this thermostable residue. A further argument in favor of the importance of glutathione is the fact that in cataract there is little or no glutathione remaining in the lens. That this diminution is not secondary to the occurrence of the opacity of the lens but is rather a part of the cataractous process itself is indicated by the observation of two of us [5] that the glutathione in the lens of an animal on a diet high in galactose may be markedly diminished before the appearance of any opacity.

From the Department of Physiological Chemistry and the Department of Ophthalmology, Northwestern University Medical School.

1. Hopkins, F. G.: Biochem. J. **15**:286, 1920.

2. Bellows, J. G., and Rosner, L.: Biochemistry of the Lens: Preparation of Glutathione from Crystalline Lens, Arch. Ophth. **16**:1001 (Dec.) 1936.

3. Adams, D. R.: Proc. Roy. Soc., London, s.B **98**:244, 1925.

4. Goldschmidt, M.: Arch. f. Ophth. **113**:160, 1924.

5. (*a*) Bellows, J. G.: Biochemistry of the Lens: The Influence of Vitamin C and Sulfhydrils on the Production of Galactose Cataract, Arch. Ophth. **16**: 762 (Nov.) 1936. (*b*) Bellows, J. G., and Rosner, L.: Am. J. Ophth. **20**:1109, 1937.

On the other hand, some investigators think that the importance of glutathione in the metabolism of the lens has been overestimated. Krause [6] has pointed out that no specific essential function has yet been found for glutathione, but he has suggested that it may have a function in the synthesis of lens protein.

In view of the interest centering about this compound, it seemed that further investigation into its variation in concentration under different physiologic conditions was indicated. Potentiometric studies [5b] on the cortex and nucleus of the lens showed a wide variation in potential between its cortical and its nuclear portion. Evidence will be presented indicating that this difference is due to a change in the concentration of glutathione. A more complete study of the concentration of glutathione in the two portions of the lens was therefore undertaken, particularly with relation to age and to the feeding of galactose.

STUDIES ON THE CONCENTRATION OF GLUTATHIONE IN THE LENS WITH RESPECT TO ITS VARIATION WITH AGE

Rats from 1 day to 14 months old were used in our experiments. The lenses were removed, and their cortical and nuclear portions separated, ground with metaphosphoric acid (2.5 per cent) and centrifuged to remove the precipitated proteins. Aliquot portions were used to determine the amount of glutathione and of ascorbic acid present. The total reducing power of the lens extract was determined by titration with potassium iodate in acid solution; the reducing power of the ascorbic acid, by titration with Tillman's reagent. The difference between the total reducing power and the reduction by ascorbic acid was attributed to glutathione. This assumption was shown to be valid by Evans.[7]

As seen from table 1, the glutathione concentration of the lens in very young rats is low compared with that of older animals. In these experiments the amount of glutathione rose rapidly from about 44 mg. per hundred grams at the age of 1 day to about 200 mg. three weeks later and reached a peak of 300 mg. per hundred grams at the age of 4 months, after which there was a slow decline.

In very young animals no differentiation could be made between the cortex and the nucleus of the lens. However, in animals 3 weeks of age and over, in which a separation between the central and the peripheral portion of the lens was possible, it was found that the concentration of glutathione was higher in the cortex than in the nucleus. This difference increased with age. Our studies indicate that the con-

6. Krause, A. C.: The Biochemistry of the Eye, Baltimore, Johns Hopkins Press, 1934.

7. Evans, E. D.: Nature, London **134**:181, 1934.

centration of glutathione in the nucleus of the lens remains relatively constant with aging of the lens, while that of the cortex varies significantly with age.

As to the ascorbic acid in the lens, it appears to be subject to a greater amount of fluctuation than does glutathione. Because of its great variability, no conclusion can be drawn at the present time.

EFFECT OF GALACTOSE ON THE CONCENTRATION OF GLUTATHIONE IN THE LENS

Since we have previously shown the amount of glutathione to be diminished when galactose is fed, we undertook to determine the influ-

TABLE 1.—*Variation in the Concentration of Glutathione and of Ascorbic Acid in the Lens with the Age of the Rat*

	Glutathione, Mg. per 100 Gm.			Ascorbic Acid, Mg. per 100 Gm.		
Age of Litter	Total	Cortex	Nucleus	Total	Cortex	Nucleus
1 day	44	...	...	9	..	..
3 days	100	...	...	10	..	..
10 days	62	...	...	15	..	..
10 days	69	...	...	57	..	..
17 days	94	...	...	14	..	..
17 days	86	...	...	12	..	..
21 days	131	153	106	32	32	31
22 days	125	...	...	11	..	..
22 days	114	156	82	28	37	23
25 days	184	...	...	..	10	..
26 days	200	236	141	11	13	10
2 months	179	371	105	13	14	10
2 months	173	371	82	14	14	13
2 months	172	371	82	13	14	12
4 months	278	497	107	15	19	12
4 months	320	516	133	19	20	18
7 months	207	341	107	12	10	14
7 months	216	339	107	10	10	10
11 months	233	434	61	13	14	12
11 months	193	316	100	13	15	11
14 months	203	285	87	5	5	5

ence of age on the rate and site of this loss, i. e., whether from the nucleus or from the cortex.

Litters of rats varying in age from 21 days to 14 months were used. The lens of one eye of each animal was removed; the lenses were pooled, and the glutathione and ascorbic acid contents were determined. The animals were then placed on a diet containing 50 per cent galactose for a given period. At the end of this time the glutathione and ascorbic acid contents were determined in the remaining lenses.

From the data in table 2, it is readily observed that the glutathione in the lens decreases in a short time when the animals are fed galactose. This decrease appears to be more marked in the cortical layer (of the lens) than in the nuclear layer. There seems to be no particular change in the rate of loss of glutathione with age. In the older lenses, because of their higher initial content, a longer period is required for the glutathione to fall to a given level. A further observation from these

data is that as the level of glutathione drops when galactose is fed the lens retains the remaining glutathione more tenaciously. Thus, during the period covered by these experiments the lowest glutathione value reached was 38 mg. per hundred grams (table 3).

To determine whether the glutathione in the lens would return to normal if the animals were replaced on a stock diet, the following experiment was performed: Two litters, one consisting of 6 and another of 8 rats, were used. The normal glutathione values were determined for 2 rats of each litter. The litters were then placed on a galactose diet for four days. The glutathione content in one eye of each remaining rat was determined, and the animals were then placed on a normal stock

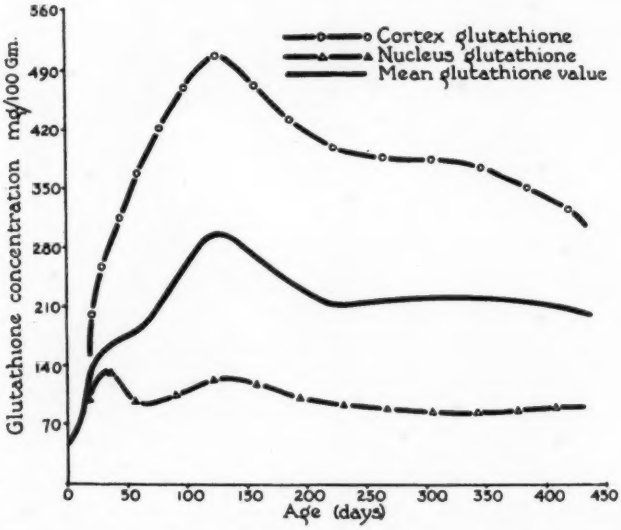

Chart 1.—Relation between age and the concentration of glutathione in the lens.

TABLE 2.—*Effect of Galactose Feeding on the Concentration of Glutathione and of Ascorbic Acid in the Lens*

Age of Litter	Days on 50% Galactose	Glutathione, Mg. per 100 Gm.						Ascorbic Acid, Mg. per 100 Gm.					
		Before Feeding			After Feeding			Before Feeding			After Feeding		
		Total	Cortex	Nucleus	Total	Cortex	Nucleus	Total	Cortex	Nucleus	Total	Cortex	Nucleus
21 days	9	131	153	106	51	32	65	32	32	31	14	14	15
22 days	4	114	156	82	89	116	56	28	37	23	16	16	15
26 days	2	200	236	141	155	152	162	11	13	10	7	6	8
2 mo.	2	179	371	105	94	123	69	11	14	10	9	7	10
2 mo.	2	173	371	82	95	123	75	14	14	13	10	7	13
2 mo.	2	172	371	82	95	123	70	13	14	11	12	7	16
4 mo.	3	278	497	107	136	221	56	13	15	11	15	19	12
4 mo.	14	320	516	133	47	49	45	19	20	18	10	10	10
7 mo.	2	207	314	107	159	244	63	12	10	13	9	8	10
7 mo.	2	216	339	107	155	215	82	10	10	10	10	10	10
11 mo.	3	233	434	61	146	249	40	13	14	12	18	21	15
11 mo.	7	198	316	100	76	128	28	13	15	11	11	9	13
14 mo.	4	208	285	87	137	150	114	5	5	5	8	7	10
14 mo.	10	...	...	...	51	65	35	..	..	..	7	7	6

diet. The glutathione values for the remaining eyes were determined eleven and seventeen days later, respectively. The results as shown in table 3 reveal that the concentration of glutathione returns to a value at least as high as that of normal animals of the same age. It is of interest to note that the usual marked difference between the values for the cortex and those for the nucleus is absent, the two portions of the lens containing almost similar amounts of glutathione.

A litter of animals with mature galactose cataract and practically no glutathione in the lens of one eye was placed on a normal stock diet for three weeks. At the end of this time one could see normal lens fibers in the periphery of the lens of the remaining eye of each rat by means of the ophthalmoscope. The lenses of these animals contained

TABLE 3.—*Effect of Galactose Feeding Followed by a Normal Diet on the Concentration of Glutathione and Ascorbic Acid in the Lens*

		Before Feeding Galactose		Four Days on Galactose		Seventeen Days on Normal Diet			
						Glutathione, Mg. per 100 Gm.		Vitamin C, Mg. per 100 Gm.	
Litter	Age, Days	Glutathione, Mg. per 100 Gm.	Vitamin C, Mg. per 100 Gm.	Glutathione, Mg. per 100 Gm.	Vitamin C, Mg. per 100 Gm.	Cortex	Nucleus	Cortex	Nucleus
1	25	184	9.8	41	5.7	211	200	15.7	19.5
2	22	125	10.8	38	5.0				
After 11 days on normal diet						251	206	8.4	8.9

TABLE 4.—*Concentration of Glutathione in Normal and Cataractous Lenses*

	Glutathione, Mg. per 100 Gm.	Vitamin C, Mg. per 100 Gm.
Central cataractous portion of lens	0*	0*
Normal peripheral portion of lens	231.0	6.0

* In this experiment concentrations of glutathione below 20 mg. per hundred grams or of vitamin C below 1.5 mg. per hundred grams were not measurable.

132 mg. of glutathione per hundred grams and 3.5 mg. of vitamin C per hundred grams. That glutathione and vitamin C are contained in the normal lens fibers laid down subsequent to the formation of the galactose cataract is shown by the determination of these substances in the cataractous and normal portions of the lenses of another litter of similarly treated rats (table 4).

This difference is strikingly brought out by the application of the nitroprusside test to a cross section of this type of lens. The normal peripheral fibers give a reddish purple hue, whereas the central cataractous portion remains unstained, indicating an absence of sulfhydryls.

THE RELATION BETWEEN GLUTATHIONE AND LENS POTENTIAL

In a previous report we presented data correlating lens potential with age and with the onset of cataract. Our interest was aroused concerning the question of what factors in the lens give rise to the lens potential

and cause its variation under certain physiologic conditions. We therefore undertook to determine the potentials of certain constituents of the lens and to reproduce the potentials found in the lens.

Before entering into a discussion of this work, we should perhaps define the term potential. When a substance may exist in both an oxidized and a reduced form (such as Fe++ and Fe+++), a solution containing this substance sets up an electrical potential at an inert electrode. This potential is a function of the relative amounts of the oxidized and the reduced form of the substance present, becoming more positive with an increase in the oxidized form and vice versa. This phenomenon is termed an oxidation-reduction potential and represents the tendency of the solution to deposit electrons on or to remove electrons from the electrode. Not all substances which exist in reduced and oxidized forms give such characteristic potentials. Glutathione, for

TABLE 5.—*Relation of the Potential to the Concentration of Beta Crystallin, Glutathione and Ascorbic Acid in a Mixture at p_H 7.30*

Substance	Concentration, Mg. per 100 Gm.	Approximate Ratio of Concentration to That Found in the Lens	Potential, Millivolts
Beta crystallin	200.0	1:85	156
Glutathione	47.4	1:7	—13*
Ascorbic acid	6.0	1:4	—11†

* The potential of glutathione plus beta crystallin.
† The potential of glutathione and beta crystallin plus ascorbic acid.

example, appears to give a potential dependent only on concentration of the reduced form. The significance of this phenomenon is still debatable.

In all of our work a gold electrode was used rather than a platinum electrode, as the former seemed to arrive at equilibrium with its surrounding medium more rapidly. It was found impossible to reproduce potentials when the system was in contact with air. A steady stream of purified nitrogen was therefore passed through the solution. This also served to keep the medium well mixed. All determinations were performed in solutions buffered with phosphates to p_H 7.3.

Considering concentration and potential activity, it appeared that of the crystalloids only glutathione and ascorbic acid might significanctly contribute to the potential. No work of this nature has previously been done with proteins, but it was reasoned that the main potential-giving group of proteins is probably the –SH group. From this point of view, beta crystallin should be potentially quite active, alpha crystallin slightly active and albuminoid active hardly at all. This line of reasoning was proved valid by experiment. The potential of a solution of alpha crystallin was determined, and beta crystallin was then added; the potential changed to the normal potential of beta crystallin. The addition of

alpha crystallin to beta crystallin produced no change in the potential. The same was true for albuminoid.

An attempt was then made to determine whether ascorbic acid, from the point of view of its small concentration in relation to glutathione and beta crystallin, could contribute significantly to the lens potential.

Table 5 illustrates the significance of beta crystallin, glutathione and ascorbic acid so far as they contribute to the potential in the lens. In this experiment each of these substances was added in the order named. Ascorbic acid, even when present in a greater quantity than either of the other two constituents, as compared to its concentration in the lens, produced no significant change in the potential given by the mixture of

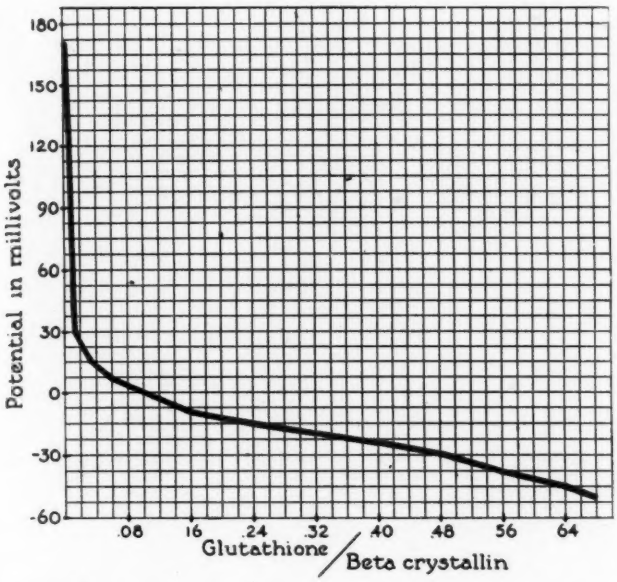

Chart 2.—Effect of the glutathione-beta crystallin ratio on the potential.

TABLE 6.—*Relation of the Potential to the Relative Concentrations of Glutathione and Beta Crystallin at* p_H *7.30*

Concentration, Mg. per 100 Gm.		Glutathione–Beta Crystallin Ratio	Potential, Millivolts
Glutathione	Beta Crystallin		
197.4	300	0.66	— 50
54.5	100	0.54	— 36
47.4	200	0.24	— 13
54.5	500	0.11	— 3
30.0	500	0.06	+ 6
14.5	500	0.029	+ 15
3.4	320	0.010	+ 31
4.0	500	0.008	+ 43
2.0	500	0.004	+140
0.0	500	.....	+170

beta crystallin and glutathione. Ascorbic acid was therefore ruled out as a factor contributing to the lens potential.

It is apparent, then, that the lens potential is determined by the relative concentrations of glutathione and beta crystallin in the lens and should be capable of duplication by in vitro mixtures of these two substances. A series of mixtures containing varying quantities of glutathione and beta crystallin were therefore prepared and their potentials determined.

Table 6 illustrates how the potential varies with variation of the ratio between concentrations of glutathione and beta crystallin. It may be noted that as the concentration of glutathione rises with respect to that of beta crystallin the potential falls. Chart 2 shows this relation strikingly. From this curve can, conversely, be estimated the ratio of glutathione to beta crystallin if the potential of the mixture is known.

COMMENT

Our findings indicate that the glutathione content of the crystalline lens at birth approximates that of blood. This observation is in agreement with the fact that in embryonal life the lens has a blood supply and in consequence has no more need for a special respiratory mechanism than has any other tissue. However, in postnatal life this blood supply is no longer available, necessitating a substitute mechanism. That glutathione takes part in this respiratory function was first indicated by Goldschmidt. Apparently in postnatal life some mechanism is set in motion (probably in the lens) that stimulates the production of glutathione. Thus there occurs a sharp rise in the glutathione content in the first few days of life to meet the increasing metabolic and growth demands of the lens. Because of the continuous requirements of the lens during its period of greatest physical development, the amount of glutathione continues to increase until the fourth month of life, after which it declines slowly.

It would seem that the concentration of glutathione in the nucleus shows no marked variation with age, the major change taking place in the cortical portion of the lens. This observation is in agreement with the fact that the nucleus of the lens maintains a sluggish metabolism as compared to the cortex. Where there is no defined nucleus, as in the very young animal, there is no large difference in the concentration of glutathione in the inner and the outer layer of the lens. This may be expected, since the lens fibers are all young with no great variation in age.

When animals are fed galactose, the glutathione in the lenses diminishes. We found that a longer time is necessary for the lenses of older animals to become depleted of this substance, but this appears

to be due to its initial higher value in the older lenses and not to a difference in rate of loss. The loss of glutathione in the cortex is rapid at the outset and becomes slower as feeding is continued. However, in the nucleus in which the concentration is initially low, glutathione is lost slowly. This difference in rate of loss can perhaps be explained as due either to the greater exposure of the cortex to external influences or to the difference in the metabolic activity of the two portions. From the latter point of view the respiratory activity of the cortex should diminish and approach that of the nucleus after several days of feeding with galactose. Measurement of the comparative oxygen uptake of the cortex and the nucleus before and after the feeding of galactose would be necessary to prove this point.

Although in young animals the appearance of cataract shortly follows the loss of glutathione, in older animals such a loss may occur long before the appearance of opacities. Any attempt at an explanation of this observation must involve discussion of the cause of glutathione depletion after the feeding of galactose. Such a loss may be explained on the basis of the phenomenon observed by two of us [8] that the permeability of the capsule of the lens decreases in the presence of galactose. It is conceivable that this decreased permeability upsets the metabolism of the lens either by preventing passage of nutrients into the lens or by inhibiting the escape of waste products from the lens. This upset in the metabolism of the lens might interfere with the production of glutathione or increase its rate of breakdown. It has been shown by Friedenwald [9] and by Gifford and his co-workers [10] that with increasing age there occurs a decreased permeability of the capsule of the lens. Since this diminution occurs slowly, the lens may become adapted to this decreased permeability and be therefore not so sensitive to further decrease. Furthermore, the smaller metabolic activity of the older lens may make it more insensitive to external influences.

It appears from our data regarding the return of glutathione to the lenses of rats placed on a normal diet after a period on a galactose diet that the lens retains its ability to recover from incipient cataract. This recovery includes a return of normal transparency to the lens fibers as observed by the ophthalmoscope and a regaining of its normal concentration of glutathione. Even after the occurrence of mature cataract, the lens can still lay down normal lens fibers in the periphery. The

8. Bellows, J. G., and Rosner, L.: Biochemistry of the Lens: XI. The Effect of Galactose on the Permeability of the Capsule of the Lens, Arch. Ophth. **20**: 80 (July) 1938.

9. Friedenwald, J. S.: Permeability of the Lens Capsule to Water, Dextrose and Other Sugars, Arch. Ophth. **4**:350 (Sept.) 1930.

10. Gifford, S. R.; Lebensohn, J. E., and Puntenny, I. S.: Biochemistry of the Lens: The Permeability of the Capsule of the Lens, Arch. Ophth. **8**:414 (Sept.) 1932.

opaque portion, however, remains a dead tissue, being unable to recover its transparency or glutathione.

We have previously presented studies dealing with variations in the potential of the cortex and nucleus of the lens under different physiologic conditions. The average potential in the cortex of the lens of young rats was found to be 28 millivolts, while that in the nucleus was 40 millivolts. In old rats, however, the cortex potential was 7 millivolts and the nucleus potential 132 millivolts. After a period of feeding the rats galactose the potential in the lenses of young rats rose to 40 and 150 millivolts in the cortex and nucleus, respectively; in the old rats the potential correspondingly rose to 42 and 153 millivolts. These results led us to attempt to find the origin of the lens potential and the factor or factors causing its variation under the physiologic circumstances mentioned. Thus, to glutathione and beta crystallin were eventually attached responsibility for the lens potential and its variations, and a curve was drawn showing the relation between the

TABLE 7.—*Comparison of Observed and Theoretic Potential in the Cortex and Nucleus*

	Cortex		Nucleus	
	Observed Potential, Millivolts	Theoretic Potential, Millivolts	Observed Potential, Millivolts	Theoretic Potential, Millivolts
Young rats	28	24	40	90
Old rats	7	20	132	120

glutathione-beta crystallin ratio and the resulting potential. If it is assumed that the beta crystallin content of the lens remains constant at 17 per cent (Krause's figure, which we may accept as a working basis), theoretic values can be obtained from the glutathione–beta crystallin ratio by interpolating in chart 2. A comparison between potentials determined with in vitro mixtures of glutathione and beta crystallin and those actually found in lenses with corresponding relative amounts of the two substances is shown in table 7. The actual and theoretic values show a good correlation in all instances except those for the nucleus of young rats. It may be pointed out that the portion of the curve concerned with this glutathione–beta crystallin ratio undergoes a marked change in potential with a slight change in this ratio. Thus, only a small error produces large variations in potentials.

This sharp change in potential with small variations in the concentration of glutathione (beta crystallin remaining constant) might in fact be utilized as a more sensitive method for the estimation of glutathione than actual chemical determination when this substance is present in small quantities.

RETINITIS PROLIFERANS

CLINICAL AND HISTOLOGIC STUDIES

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CHICAGO

The term retinitis proliferans is purely descriptive and applies to a variety of pathologic conditions due to different causes. The expression retinitis designates a pathologic process in the retina, not necessarily of an inflammatory nature, and the adjective proliferans indicates the plastic character of the lesions.

Clinical and histologic studies of eyes during the earlier stages of proliferating retinitis reveal that there are two principles involved in the production of this condition, leading to two essentially different types of retinitis proliferans: type 1, in which the formation of connective tissue precedes the formation of new blood vessels, and type 2, in which there is a primary formation of new vessels with secondary production of a scaffolding of delicate connective tissue.

In type 1 exudation or hemorrhage from the retina into the vitreous, caused by inflammatory or traumatic alterations of the retinal vessels, is the primary event, and organization of the extravasation leads to the formation of the well known strands and membranes.

The group of conditions producing this type of proliferating retinitis includes: (1) tuberculous retinal periphlebitis or recurrent hemorrhages into the vitreous in juvenile subjects, (2) neuroretinitis papulosa of the secondary stage of syphilis, (3) all injuries of the retinal vessels followed by hemorrhage and (4) secondary involvement of the inner retinal surface in severe cases of uveal inflammation, as recently described clinically by Arnold Knapp.[1] In the last-mentioned condition exudate originating elsewhere in the eye is deposited on the inner retinal surface and there may produce alterations, which lead finally to partial organization of the deposit. These alterations will be discussed in a future publication.

In type 2 the main factor in the production of the plastic lesions is a slow circulatory impairment due to degenerative vascular disease, with the formation of new compensating anastomotic channels, with or without hemorrhages at first. The retinitis is produced mainly by a disturbance of the venous circulation due to sclerotic changes of neighboring structures and preexisting unfavorable topographic anatomic relationship.

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1. Knapp, A.: Formation of Preretinal Connective Tissue in the Vitreous in Acute Choroiditis: Report of Three Cases, Arch. Ophth. **18**:558 (Oct.) 1937.

This type of proliferating retinitis is often found in association with (1) diabetes, (2) syphilis of the tertiary stage with predominating endarteritis or (3) arteriosclerosis, including forms in which there is occlusion of the central retinal vein.

To illustrate the two types of retinitis proliferans, several eyes are described in which typical examples of each type were present.

Clinical examination of a patient 43 years of age, with a healed neuroretinitis papulosa of the right eye and reduction of vision in the affected eye to perception of hand movements, revealed proliferating retinitis of type 1. The Wassermann reaction of the blood, which had never been tested before, was strongly positive. The results of other examinations were irrelevant, and the other eye was normal. The optic disk in the right fundus was ill defined, pale and partly covered with connective tissue, indicative of postneuritic atrophy of the optic nerve. Connective tissue also accompanied some of the large retinal veins, and fanlike structures spread over several old chorioretinitic lesions; connective tissue was found in all those places where the inflammatory process had been located, namely, at the site of the papular lesions, along the retinal veins and on the nerve head, thus indicating the characteristic trio of neuritis, periphlebitis and disseminated chorioretinitis, which was first described by A. Fuchs,[2] who also named this condition occurring in the secondary papular stage of syphilis, neuroretinitis papulosa.

The connective tissue forming these strands and membranes was dense and white and did not contain visible newly formed blood vessels (fig. 1).

For histologic demonstration of the first type of retinitis proliferans, two eyes with tuberculous retinal periphlebitis, one in the early and one in the late stage, appeared suitable. The first stage of this disease is well known clinically and histologically. The investigations of Axenfeld,[3] Fleischer[4] and Wolf[5] are responsible for the knowledge of its tuberculous nature; however, the appearance is more that of nonspecific, proliferative inflammatory changes on an allergic basis than of typical tuberculous granulation tissue. The disease manifests itself

2. Fuchs, A.: Ueber einige seltene luetische Erkrankungen des Auges, Ztschr. f. Augenh. **59**:213, 1926.

3. Axenfeld, T., and Stock, W.: Ueber die Bedeutung der Tuberkulose in der Aetiologie der intraokularen Hämorrhagien, und der proliferierenden Veränderungen in der Netzhaut, besonders über Periphlebitis retinalis bei Tuberkulösen, Klin. Monatsbl. f. Augenh. **49**:28, 1911.

4. Fleischer, B.: Die juvenile Periphlebitis retinae, mit ihren Folgeerscheinungen; eine echte Gefässtuberkulose der Netzhaut, Klin. Monatsbl. f. Augenh. **17**: 769, 1914.

5. Wolf, H.: Zur Angiopathia juvenilis mit Bemerkungen über den Bau und das Wesen der Limitans interna retinae, Arch. f. Augenh. **89**:54, 1921.

first in the lymph spaces around the venous branches. Dense round cell infiltration distends the perivascular lymph spaces, thus forming nodules and spindle-shaped thickenings of the veins, which gradually encroach on and destroy the walls of the veins. The resulting intraretinal and preretinal hemorrhages and exudates are partly absorbed and partly organized. When the hemorrhages are extensive, and particularly when they are recurrent, capillaries but rarely large blood vessels grow into the organizing extravasation.

Fig. 1.—Retinitis proliferans of type 1 associated with neuroretinitis papulosa.

In figure 2 such an advanced stage of recurring hemorrhages in the vitreous of a juvenile subject is shown. The retina and optic nerve are covered with a dense layer of connective tissue. This thick membrane is rich in cells and capillaries, and the older layers show stratification.

Clinical examination of a diabetic patient aged 46 revealed retinitis proliferans of type 2. The patient had been under observation in the ophthalmic clinic of Rush Medical College for a number of years, and the earliest stage of proliferating retinitis was observed in the

better eye. When first seen, this patient had been on diabetic management for four years, and dietary measures sufficed to eliminate glycosuria. The blood pressure was 134 systolic and 66 diastolic. Vision in the right eye was first noted to fail three years prior to the first examination, while that in the left eye was first noted to fail three weeks prior to this examination, at which time the corrected vision in the right eye was 0.1 and that in the left eye was 0.4.

In the right fundus there was a large fan-shaped membrane (fig. 3) of delicate, semitransparent connective tissue, which contained a great number of large and small newly formed blood vessels in a brushlike

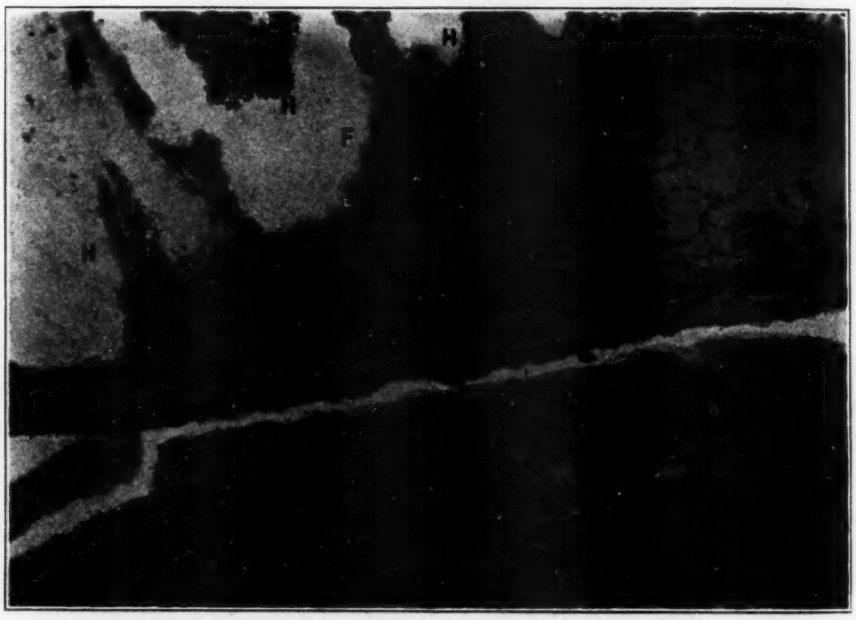

Fig. 2.—Retinitis proliferans of type 1 associated with a late stage of tuberculous periphlebitis. *H* indicates free hemorrhages in the vitreous; *F*, fibroblasts, and *C*, capillaries.

arrangement. The membrane completely covered the optic disk, where it appeared to have its base. Along the large retinal vessels and in the macular area there were numerous fatty degenerative retinal lesions and hemorrhages, mostly of the small capillary type.

In the left fundus (fig. 4) a small brushlike arrangement of newly formed vessels, arising from the inferior nasal vein near the border of the optic disk, proceeded straight forward into the vitreous. There was no noticeable connective tissue between these blood vessels at first. There was only mild retinal angiosclerosis visible in each eye. During the following six months of observation the brushlike arrangement of

newly formed vessels became larger (fig. 5), and delicate grayish connective tissue became visible between the vessels. The proliferating retinitis in this eye began to resemble that of the right eye (fig. 3).

This type of retinitis proliferans was also seen clinically in the left eye (fig. 6) of a 64 year old woman, who came to the ophthalmic clinic of the Billings Memorial Hospital, complaining of failing vision in the left eye of four years' duration. The corrected vision in the right eye was 0.8 and that in the left eye was 0.6 — 3. There was moderate bilateral retinal angiosclerosis with obliterating endarteritis evident in some small branches in each eye. In the left fundus several

Fig. 3.—Advanced retinitis proliferans of type 2 associated with diabetes.

brushlike arrangements of new vessels, surrounded by delicate connective tissue and ending with coil-like convolutions in the vitreous, were attached to the inferior nasal quadrant of the disk.

Medical examination revealed moderate arterial hypertension of the benign essential type and no other pathologic changes.

The predominating characteristic of the proliferating retinitis in these eyes was the great number of newly formed vessels, which were partly present in a brushlike arrangement, especially in the initial stage, and originated from the nerve head. The connective tissue between had a delicate, semitransparent appearance and thus a less white color than that in the eyes previously studied.

It is difficult to illustrate histologically the early stage of the second type of retinitis proliferans, but it is possible to distinguish its char-

acteristics in more advanced conditions. The first eye showing this type of retinitis proliferans to be considered here was the left eye of a diabetic patient 63 years old, who had at the time of the enucleation a blood pressure of 210 systolic and 120 diastolic. The eye was enucleated ten weeks after obstruction of the central retinal vein and eight weeks after onset of the secondary glaucoma. Examination of the fundi a year and a half before revealed marked retinal angiosclerosis, numerous, mostly capillary hemorrhages scattered over both posterior polar regions and groups of fatty degenerative lesions around the left disk and macula. Both disks were normal.

Most striking was the histologic appearance of the retinal arteries. The central artery and its immediate branches were filled almost com-

Figure 4

Figure 5

Fig. 4.—Very incipient retinitis proliferans of type 2 associated with diabetes.
Fig. 5.—The same eye shown in figure 4 six months later.

pletely with endothelial and subendothelial proliferation. This, together with some elastic lamellation and considerable thickening of the tunica media and the perivascular sheaths, transformed the central artery into a rigid tube, which compressed the central vein within the lamina cribrosa. There were large intraretinal hemorrhages along the course of the engorged veins. The lower part of the optic nerve and the adjoining retina were covered with a delicate membrane, which contained newly formed, thin-walled blood vessels in cross and longitudinal sections, some of them of large caliber (fig. 7). The framework of the membrane around these vessels was extremely delicate, consisting of a few fibroblasts, wandering cells and a faintly staining, almost homogeneous transudate. These newly formed structures were on the inside of the internal limiting membrane.

The place where the internal limiting membrane is perforated by the newly formed blood vessels can usually be found in serial sections (fig. 8). Only when the attempts at anastomosis originate from the nerve head the newly formed vessels do not have to perforate the internal limiting membrane and thus have a more straightforward course, projecting into the vitreous rather than creeping along the inner surface of the retina.

The considerable size of the new blood vessels and the marked degree of retinal angiosclerosis in this case, which had caused considerable arteriovenous compression even a year and a half prior to the

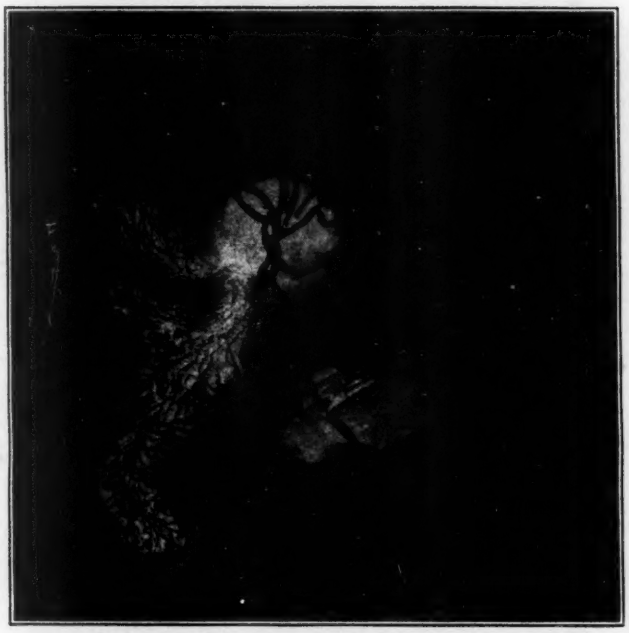

Fig. 6.—Retinitis proliferans of type 2 associated with arteriosclerosis.

occlusion of the central vein, make it probable that the proliferating processes were well under way before the complete venous obstruction, which occurred only ten weeks prior to enucleation.

The second eye showing retinitis proliferans of type 2 was the left eye of a 68 year old woman which was enucleated on account of secondary glaucoma two years after occlusion of the central retinal vein.

The optic nerve was deeply excavated and there was a dense network of tortuous, partly preformed but dilated and partly newly formed veins in the retrolaminar and prelaminar regions. From some of the veins in the prelaminar region there arose branches, which could be followed far into the vitreous, where some of them split up into a brushlike arrangement of smaller vessels (fig. 9); others took on a

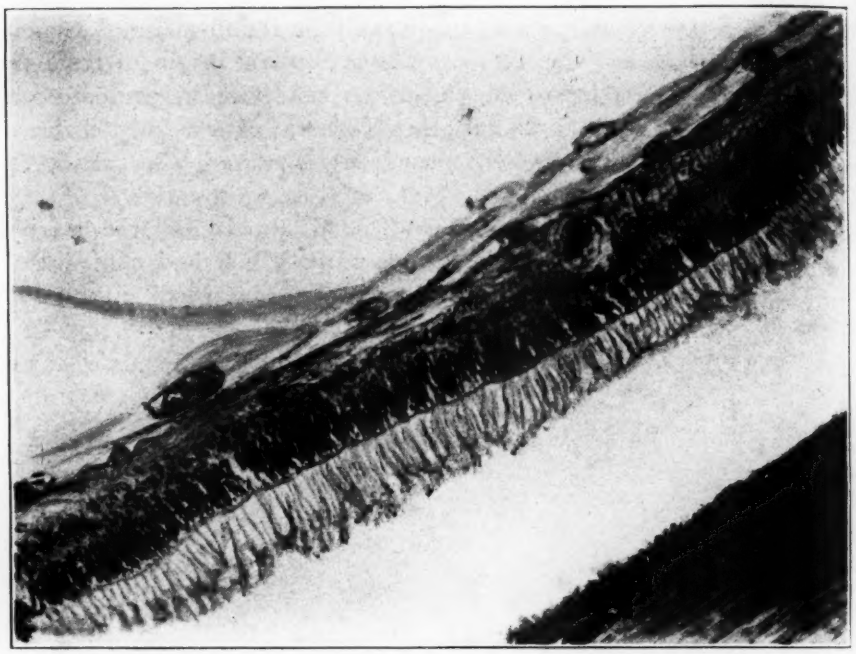

Fig. 7.—Earlier stage of retinitis proliferans of type 2. Large newly formed blood vessels and delicate connective tissue on the inside of the retina are present.

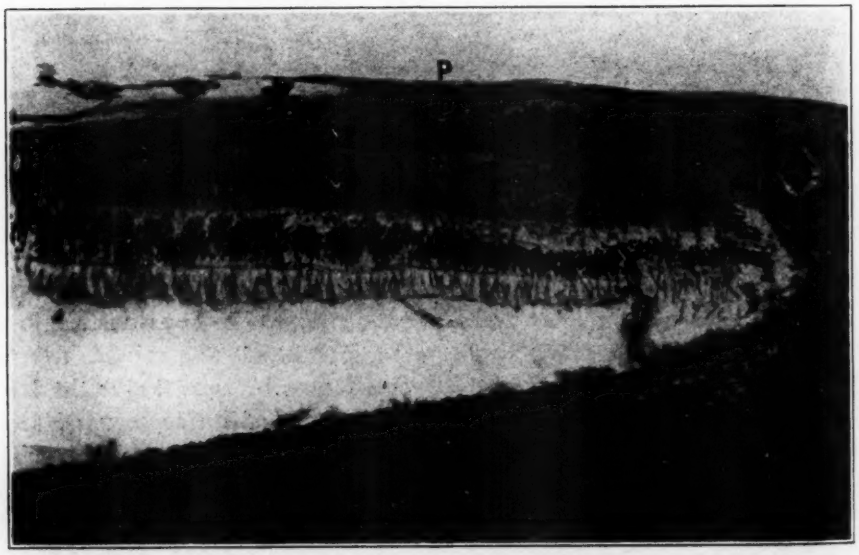

Fig. 8.—Section of the same eye as shown in figure 7. The internal limiting membrane is perforated by newly formed vessels. *P* indicates the point of perforation.

Fig. 9.—Late stage of retinitis proliferans of type 2. The blood vessel in the vitreous is split up into a brushlike arrangement of small vessels. *V* indicates a vein in the nerve head; *H*, hyaline degeneration of connective tissue, and *B*, a brushlike arrangement of new capillaries.

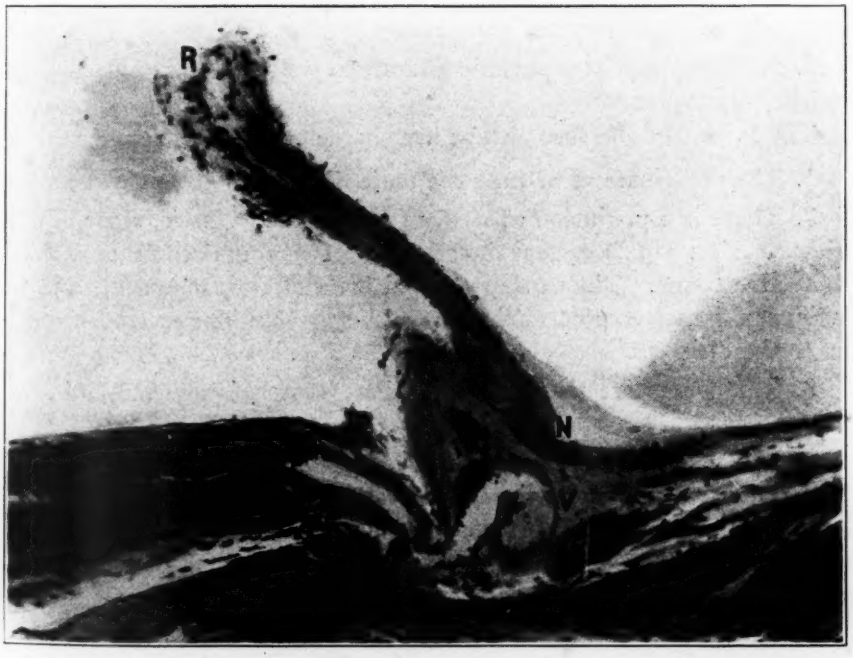

Fig. 10.—Late stage of retinitis proliferans of type 2. Large newly formed blood vessels are seen in the vitreous with a recurrent course. *V* indicates a dilated and tortuous vein in the nerve head; *R*, a recurrent newly formed vein in the vitreous, and *N*, neuroglia.

recurrent course, running backward again (fig. 10). Free hemorrhages around some of these thin-walled vessels were in the process of organization.

In this late stage the framework of the strands carrying the new vessels was denser, consisting of proliferated fibroblasts, neuroglia and a dense homogeneous material, which was shown to be hyaline by the Van Gieson stain. But even in this late stage, the amount of connective tissue was negligible compared with the amount of newly formed blood vessels.

SUMMARY AND CONCLUSION

On the basis of clinical and histologic studies it is possible to distinguish two types of retinitis proliferans and to deduce, in a not too advanced stage, from the location and appearance of the plastic lesions the nature of the primary disturbance.

The lesions of the first type are characterized as follows:

1. They may be located anywhere in the fundus.

2. They consist of dense, opaque, white or grayish-white membranes.

3. They contain no newly formed blood vessels, or only a limited number, which are rarely of large size and are seen only in the advanced stage of the disease.

4. Histologically, the connective tissue is rich in cells and not infrequently has a stratified appearance.

5. Inflammatory or traumatic alterations of the retinal blood vessels are the primary cause.

The lesions of the second type are characterized as follows:

1. They originate at or near the optic disk.

2. There is a profusion of newly formed blood vessels, some of considerable size, which appear in advance of any noticeable amount of connective tissue. They often show a brushlike arrangement, and the endings in the vitreous form either coil-like convolutions or loops, returning to the disk.

3. The connective tissue between these vessels is delicate and semitransparent.

4. Histologically, numbers of new veins can be traced to branches of the central vein in or near the nerve head. In the earlier stages there is a delicate network of fibroblasts within a homogeneous transudate between them. Later, fibroblasts and neuroglia proliferate within these strands, and hemorrhages from the thin-walled new vessels become organized and give them a more substantial appearance.

5. Degenerative vascular disease leading to circulatory impairment and venous anastomosis is the etiologic factor.

STUDIES OF THE VISUAL FIELDS IN CASES OF VERIFIED TUMOR OF THE BRAIN

DANIEL KRAVITZ, M.D.
BROOKLYN

The value of studies of the visual fields as an aid in the diagnosis of obscure conditions of the eye and brain has long been known. MacKenzie,[1] in his textbook on ophthalmology, published in 1854, called attention to the importance of hemianopic defects in the visual fields in the localization of diseases of the brain. About this time the ophthalmoscope made its appearance, and ophthalmology was placed on a firm scientific basis.

The importance of perimetry in the study of lesions that were not visible with the ophthalmoscope immediately became apparent. Troncoso [2] stated that von Graefe stressed this fact in 1856. Wilbrand [3] in 1881 reported a large number of cases and stressed the importance of examinations of the visual fields as an aid in localizing intracranial disorders. He probably was the first to see the value of using small test objects in bringing out defects that had been missed by the cruder methods then in use. In 1882 Berry [4] foresaw the importance of studies of the visual field in the diagnosis and prognosis of diseases of intracranial origin.

It was not, however, until Bjerrum and Rönne [5] did their epoch-making work on glaucoma in the early part of the twentieth century that the modern method of perimetry came into being. This method, applied by Cushing [6] and his co-workers, Heuer and Walker,[7] in analyzing defects due to chiasmal syndromes, rapidly advanced the

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Read before the Brooklyn Neurological Society, Dec. 22, 1937.

1. MacKenzie, W.: A Practical Treatise on the Diseases of the Eye, ed. 4, London, Longman [and others], 1854.

2. Troncoso, M. V.: Internal Diseases of the Eye, Philadelphia, F. A. Davis Company, 1937, p. 81.

3. Wilbrand, H.: Ueber Hemianopsie und ihr Verhältnis zur topischen Diagnose der Gehirnkrankheiten, Berlin, A. Hirschwald, 1881, p. 197.

4. Berry, G. W.: On Central Amblyopia, Ophth. Hosp. Rep., London **10**: 44, 1880.

5. Rönne, H.: Ueber das Gesichtsfeld beim Glaukom, Klin. Monatsbl. f. Augenh. **47**:12, 1909; numerous later publications.

6. Cushing, H.: The Pituitary Body and Its Disorders, Philadelphia, J. B. Lippincott Company, 1912.

7. Walker, C. B.: Some New Instruments for Measuring Visual-Field Defects, Arch. Ophth. **42**:577, 1913.

knowledge concerning defects in the visual fields in relation to tumor of the brain. Perimetric technic has been further refined by Evans,[8] so that minute circulatory defects can be detected.

There have been a great number of papers and books on the value of perimetric study in the diagnosis of intracranial tumors. It would therefore seem that one should apologize for presenting a subject so thoroughly discussed. However, as will be shown even by the small series of cases presented here, through neglect tumors of the brain are still allowed to progress until the tumor becomes inoperable or blindness supervenes.

In this paper are reports of 23 cases of verified tumors of the brain. All but 2 of the patients were seen by me preoperatively. One was seen prior to a second operation and the other after removal of the tumor. The reports of both of these cases are included because of

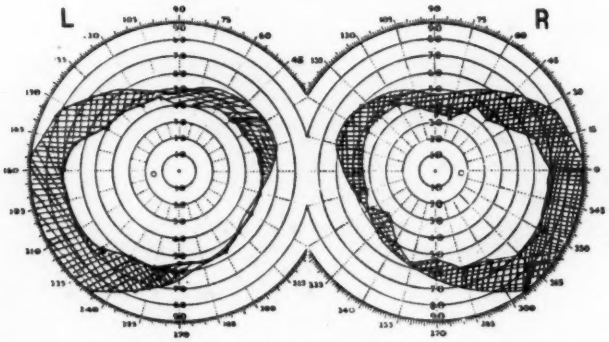

Fig. 1 (case 1).—Visual fields of F. S., taken on May 7, 1932, with a 2 mm. white test object at 330 mm.

their great clinical interest. An attempt will be made to compare the value of visual examinations of the fields with the other methods used in the diagnosis of tumor of the brain.

GROUP 1: TUMORS OF FRONTAL LOBE

CASE 1.—F. S., a man aged 34, was seen on May 2, 1932, with a history of severe pains in the forehead and vomiting for the past seven weeks. Five weeks before he went to an otolaryngologist, who advised removal of a deviated septum. As relief was not obtained by this procedure tonsillectomy was advised.

Vision was 20/30 in the right eye and 20/25 in the left eye. Improvement was not obtained with glasses. There was no nystagmus or ocular palsy. The right disk was swollen about 2.5 D., and there were many hemorrhages around and for some distance away from the disk. The left disk showed only 1 D. of swelling and appeared somewhat atrophic. The visual fields on several examinations showed an increasing concentric contraction, without localizing signs. There were no personality changes.

8. Evans, J. N.: Angioscotometry, Am. J. Ophth. **9**:489, 1926.

In view of these findings, it was felt that the patient had a tumor of the frontal lobe, and he was referred to the Brooklyn Hospital, where the following observations were made by Dr. E. J. Browder: The patient had slight difficulty in walking, with a tendency to stumble, although he never fell. There were no character changes or forced grasping. On May 11, while at the hospital, he had an attack of mental confusion and was found with his feet on the window sill, as if to climb out. Later, he remembered nothing of this episode. This was thought to be characteristic of a tumor of the frontal lobe.

On June 19 a bone flap was elevated in the right frontal region and a cyst, the size of a golf ball, was found in the right frontal lobe. It was evacuated, and the walls were cauterized. Examination proved it to be a glioma.

This case is interesting in that there was atrophy of the optic nerve on the side opposite to the site of the tumor—a paradoxic Foster Kennedy syndrome.

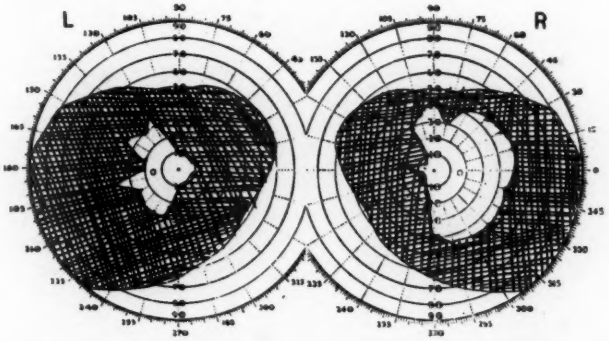

Fig. 2 (case 2).—Visual fields of C. M., taken on April 4, 1935, with a 10 mm. white test object at 250 mm. for the left eye and a 2 mm. white test object at 250 mm. for the right eye.

CASE 2.—C. M., a woman aged 29, was admitted to the Brooklyn Hospital on April 1, 1935, with a history of intermittent headaches of ten years' duration. For the past six months her vision had become progressively more blurred, and her sense of smell had been lost. She had a constant buzzing in the left ear for the past six months.

Roentgenographic examination on April 2 showed an area of lessened density of the frontal bone which was almost centrally placed a little above the orbits. The sella turcica was not well defined. It was definite that it was enlarged. The floor was depressed, probably actually eroded, and the posterior clinoid processes were eroded. In view of these findings it was thought that a tumor of the sella turcia or of the area around it and a tumor of the frontal bone were present. The growth in the frontal bone was probably metastatic.

Ocular examination on April 4 disclosed no ocular palsy and no nystagmus. The pupils reacted to light and in accommodation. There was 5 or 6 D. of swelling of both disks. There were a few hemorrhages around the left disk but none around the right. Vision in the left eye was so poor that a 10 mm. test object was the smallest that could be used. This showed a complete loss of the nasal field with retention of a small island in the temporal field. The right visual field

could be plotted with a 2 mm. test object and showed a marked concentric contraction with a loss of the nasal field. There was no evidence of secondary atrophy of either disk.

On the basis of the results of the ocular examination, it was thought that the condition was tumor of the left side of the brain, near the chiasm. For the degree of intracranial pressure and the headaches complained of by the patient, she was markedly euphoric and jocular. There was a distinct forward bulge in the middle of the forehead.

The neurologist found no signs of localizing value and was of the opinion that the condition was a tumor of the frontal lobe.

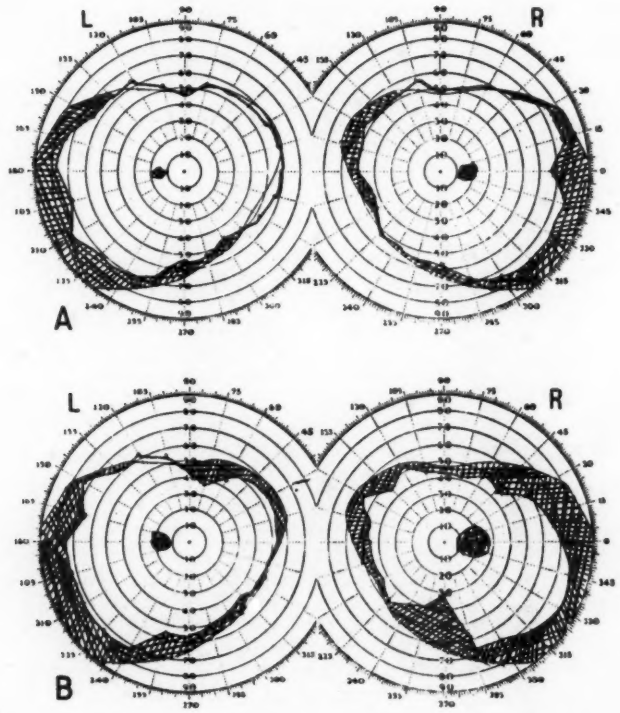

Fig. 3 (case 3).—*A*, visual fields of M. E., taken on March 9, 1933, with a 1 mm. white test object at 250 mm. *B*, visual fields of the same patient, taken on March 16, with a 1 mm. white test object at 250 mm. Vision was 13/15 in each eye.

On April 11 Dr. E. J. Browder turned down a bone flap in the right frontal region and disclosed a meningioma, the size of a small orange, occupying the frontal aspect of both anterior cranial fossae. This well defined meningioma of the midline was removed.

The loss of both nasal fields is interesting and can be explained by a flattening of the chiasm against both internal carotid arteries.

Case 3.—M. E., a woman aged 36, was admitted to the Brooklyn Hospital on Feb. 27, 1933, with a history of blurred vision and headaches for the past seven or

eight months. She has been perfectly well previously. Her illness began with a severe headache, which woke her early in the morning and lasted all day. Since then the headaches had recurred every two or three weeks. They were diffuse all over the head. Her vision had been continuously blurred since the onset. She never had diplopia. Two months before admission to the hospital she began to be annoyed by bad odors and a bad taste in the mouth. At times she would talk incoherently for a few minutes. During these attacks she was conscious but could not recall what she said. She had no dreams, hallucinations or delusions.

Neurologic examination on the day of admission revealed no apparent abnormality in the motor system. Coordination and muscle strength were good. Sensory examination gave negative results, and the cranial nerves were normal except for a questionable weakness of the right facial nerve.

In view of the history of uncinate attacks, asymmetry of the lower right side of the face, an increased knee jerk on the right and ankle jerk on the left and a history of loss of consciousness, it was thought that the patient probably had a tumor of the left temporosphenoid region.

Ocular examination on March 9 disclosed vision of 13/13 in each eye. No ocular palsy nystagmus was present. Both disks were blurred and edematous for about 1 to 1.5 D. There were no hemorrhages or exudates. The visual fields were uniformaly contracted, with a slightly greater involvement of the right nasal field. The blindspot in the right eye was much larger than in the left eye.

The results of the ocular examination, the contracted fields, with more marked involvement of the right nasal field and greater enlargement of the blindspot in the right eye, were suggestive of a tumor of the right frontal lobe.

Another survey of the eyes, on March 16, showed the picture of the fundi to be unchanged. The right visual field was further contracted, and there was a beginning defect in the lower nasal quadrant. The blindspot in the right eye showed further enlargement. The left visual field showed only a slightly increased concentric contraction.

The beginning sector-like defect observed at this examination, with the greater increased concentric contraction and enlarging blindspot on the right, was suggestive of a lesion of the right frontal lobe rather than of a lesion on the left side.

On March 22 air was injected into each ventricle. The roentgenogram showed no findings of localizing value. In view of this negative report and the uncertain neurologic signs, the patient was discharged for further observation.

On April 3, 1934, she was admitted to the Neurological Institute of New York with the following symptoms: absence of the upper abdominal reflexes, weakness of plantar flexion on each side, weakness of the muscles of the left eye, complete anosmia, pallor of the right disk and high papilledema of the left eye, bilateral paresis of the abducens nerve and facetiousness and irresponsibility.

The right visual field showed perception of light in the lower temporal and nasal quadrants only. The left visual field showed supramedial and inferolateral quadrantanopia.

A roentgenogram of the skull showed distinct evidence of an intracranial pathologic process, probably a tumor. The optic foramens were normal.

The preoperative diagnosis was meningioma of the olfactory groove.

On April 13 a right transfrontal osteoplastic flap was turned down, and a meningioma was found in the right anterior and middle fossa, which was subtotally removed. More tissue was removed on April 30 and May 19.

The postoperative diagnosis was meningioma of the right sphenoid ridge.

Of interest were the symptoms usually associated with tumor of the temporosphenoid region and the negative encephalographic picture. From a diagnostic point of view, one must evaluate the importance of an enlarging blindspot in the absence of a greater degree of choked disk on the same side. This is the first stage of the Foster Kennedy syndrome. It was possible in this case to follow the formation of the syndrome from the beginning.

Case 4.—E. R., a man aged 29, was seen on May 7, 1935, with a history that in March 1934 he began to have convulsions accompanied by immediate loss of consciousness and biting of the tongue. There was no aura or incontinence. The patient was mentally clear after the attacks, most of which occurred during the night. After April 1934 he had severe headaches, but he never vomited. Neurologic examination gave negative results, except for a doubtful weakness of the lower right side of the face and a more active ankle jerk on the right than on the left. Recently he had been having personality changes and he appeared euphoric. He was unusually sleepy. The neurologic diagnosis was tumor of the frontal lobe.

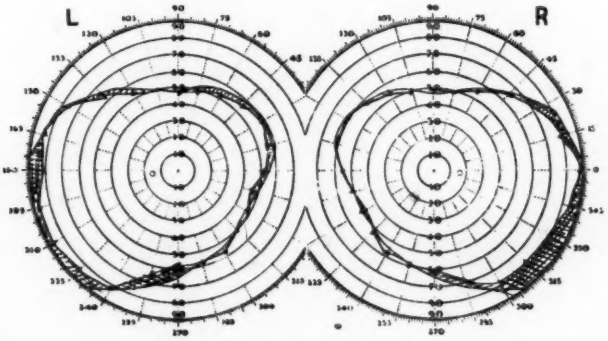

Fig. 4 (case 4).—Visual fields of E. R., taken on May 7, 1935, with a 2 mm. white test object at 250 mm.

Ocular examination disclosed vision of 20/25 in each eye. There was no nystagmus or ocular palsy. The pupils were equal and regular and reacted to light and in accommodation. The right disk was well outlined. There was an increase in the size and number of the capillaries, which gave the disk a violaceous hue. Both the arteries and the veins seemed increased in size, and there was a small flame-shaped hemorrhage near the nasal side of the disk. The left disk was somewhat more obscure, but there was no measurable edema. There were many flame-shaped hemorrhages near the disk. The visual fields were full.

The patient went to the New York Post-Graduate Medical School and Hospital, where roentgenographic examination revealed thinning of the bone over the premotor area, more marked on the left. The sella turcica was flattened from above down, and the posterior clinoid processes were eroded. There were three calcified plaques just to the right of the midline.

A diagnosis of tumor of the left premotor area was made.

Examination of the visual fields repeated at the hospital revealed no defects. On May 13 ventriculography was attempted, without success. Two days later the neurologist noted a definite weakness of the right side of the face and an exagger-

ation of the achilles tendon reflex on the right. There was partial aphasia. The patient repeated words and syllables. Examination of the fundi showed about 5 or 6 D. of swelling of both disks, and vision had depreciated to the perception of large objects. On May 16 Dr. Scarf made a ventriculogram, which showed marked distortion of the ventricles by a tumor occupying the premotor areas.

A diagnosis of parasagittal meningioma, more to the left, was made.

Craniotomy was done on the left side, and a parasagittal meningioma was disclosed which apparently arose from the falx. Subtotal removal was done. The tumor measured from 4 to 5 cm. long and from 1 to 2 cm. wide. Dr. Scarf declared it to be the largest meningioma he had ever seen.

It is amazing to see the tremendous size a slow-growing tumor may reach before giving rise to serious symptoms, but how rapidly it progresses once it has started.

CASE 5.—F. F., a man aged 47, was referred to the ophthalmic clinic of the Brooklyn Hospital because of dizziness for the past year. He had been under

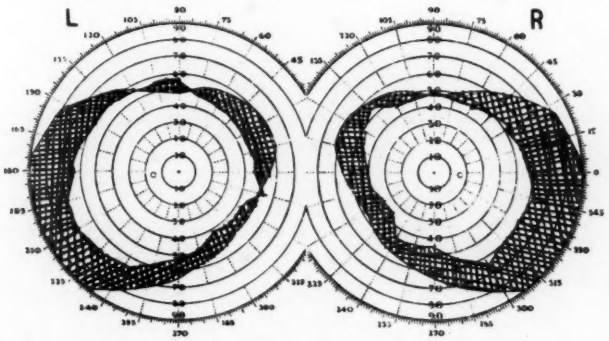

Fig. 5 (case 5).—Visual fields of F. F., taken on March 2, 1938, with a 2 mm. white test object. Vision in each eye was 16/30.

treatment at a medical clinic for three months for hypertension and obesity. His blood pressure was 158 systolic and 112 diastolic.

About a year before admission to the hospital he was discharged from the post-office, where he was a laborer, for disobedience to his superiors. About this time paralysis of the left side of the body developed. For the past year there had been a progressive deterioration in his mentality as well as of his character. In 1917 he was struck on the head by a heavy package and was knocked unconscious for a short while. In 1935 he was struck by an automobile and was again knocked unconscious. About six years before admission to the hospital he had a seizure while combing his hair. This was characterized by a ten minute period of staring and speechlessness. There were no convulsive movements, but he kept repeating "la, la, la, la." He was able to understand spoken words and nodded his head in response. These attacks had recurred about eight to ten times a year, the last taking place about five days before admission to the hospital. This time the head tilted to the right, and he vomited a little.

His complaint on admission was that he became dizzy when he stooped and that he frequently had a pain in the left side of his head. When he stretched his arms upward, he had pain back of each ear.

Vision was 16/30 in each eye. There was no ocular palsy or nystagmus. There was about 3 D. of choking of the right disk, with a small hemorrhage on the disk. The veins were dilated, and the arteries were narrow. The left disk was swollen about 2 D., and no hemorrhages were visible. There was marked secondary atrophy of both disks.

The visual fields were difficult to take and were not completely reliable, but apparently they showed nothing more than a concentric contraction, more marked in the right field.

The impression on the basis of these findings was that the patient had a tumor of either the frontotemporal or the frontoparietal region. Because of the paresis on the left side, the growth was thought to be located on the right. The patient was admitted to the Neurological Institute of the Brooklyn Hospital on March 8, 1938. The salient points of the neurologic examination were as follows: (1) complete anosmia, (2) slight facial paresis on the left side, (3) motor weakness of the left arm and leg, (4) ataxia of the left hand with the finger to nose test and (5) standing on a wide base.

The unanimous opinion of the members of the neurologic staff as well as of those of the neurosurgical staff was that the condition was a tumor of the frontal lobe on the right side. The tumor was probably a meningioma arising from the falx.

On March 14 a ventriculogram showed marked displacement of the ventricular system to the right, with the left lateral ventricle being pushed downward and medially. A lateral view showed the left lateral ventricle to be occluded in the midparietal region.

In view of the ventriculographic picture, it was thought that there was a tumor in the left frontal region.

Operation by Dr. E. J. Browder disclosed a tremendously large meningioma (from 6 to 7 cm. at its greatest diameter) embedded in the left premotor area. It was attached to the falx, from which it derived a considerable vascular supply.

The visual fields, rightfully, showed no localizing defects and so could be considered correct. However, paresis on the same side as the tumor certainly was misleading, and if it had not been for the usual carefulness shown by Dr. Browder in making a ventriculogram, in the face of definite localizing signs, much unnecessary surgical intervention would have been carried out.

CASE 6.—W. L., a man aged 24, came to the dispensary of the Brooklyn Hospital on Dec. 16, 1937, with a history of recurrent convulsive attacks for the past three months. These attacks were preceded by a slight feeling of numbness in the hands, which spread over the entire body, so that he grabbed onto the nearest object for support. Then a typical jacksonian convulsion developed, which started with the left hand and spread up that arm; the mouth was drawn to the left, followed by the turning of the head and eyes to the left. Generalized clonic convulsions developed during which consciousness was lost. Over the period of three months he had three major and four minor attacks, the longest lasting thirty minutes.

For the past year his mother had noticed a carelessness in his dress and some mental slowness. Every month or two he had attacks of severe frontal headaches, but for the past few months the headaches had been mainly in the occipital region.

There had also been episodes of momentary blindness when he arose from a sitting position. For the past four or five weeks he had been voiding involuntarily. He never vomited.

The patient was uncooperative, so that all ocular examinations were conducted with great difficulty. Vision was 20/40 + in the right eye and 20/25 + in the left eye. The pupils were equal and regular and reacted to light and in accommodation. There was no ocular palsy or nystagmus. The disks were swollen slightly more than 4 D. The visual fields showed a concentric contraction, that in the left being most marked, and there was a tendency toward a lower right quadrantanopia.

In view of the results of the ocular examination, it was thought the patient had a tumor of the left frontoparietal region. He was admitted to the Neurological Institute of the Brooklyn Hospital on Jan. 3, 1938, where the following data were recorded: The patient was untidy and unkempt and was unable to concentrate on any one subject. He answered questions sluggishly. There was impairment of judgment, and he frequently made mistakes in reading.

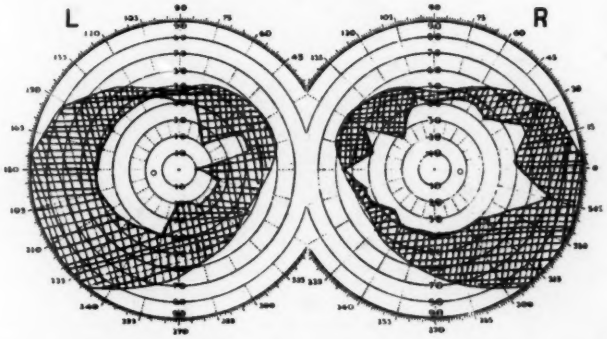

Fig. 6 (case 6).—Visual fields of W. L., taken on Dec. 16, 1937, with a 2 mm. white test object at 250 mm. Vision was 20/50 + in the left eye and 20/40 + in the right eye.

Neurologic examination showed (1) bilateral impairment of the sense of smell, (2) fine tremors of the extremities and lips, (3) deep reflexes which were slightly more active on the left and (4) good coordination. There was no stereopsis.

On the basis of the results of the neurologic examination, it was thought that there was a tumor of the right frontoparietal region, affecting secondarily, by pressure, the circulation on the left side.

A flat roentgenogram taken on January 4 showed fine mottled calcareous shadows in the right parietal area just above the temporal region. The sella turcica was apparently enlarged.

On the basis of the roentgenographic picture, it was thought that a tumor of the right parietal area was present.

The ventriculographic diagnosis was tumor in the right parietofrontal region.

On January 7 Dr. E. J. Browder turned down a bone flap in the right frontoparietal region and uncovered a highly vascularized type of malignant tumor (glioma) occupying the right premotor area and extending forward and possibly deep to involve the corpus callosum.

In 1911 Foster Kennedy [9] stressed the localizing value of the syndrome in which there is unilateral loss of vision with secondary atrophy of the optic nerve and swelling of the opposite disk. In 10 of 14 cases of tumors of the basal portion of the frontal lobe Lillie [10] found a central scotoma to be present. The scotoma was always on the side of the tumor. In 3 of the remaining cases homolateral blindness or near blindness was present. In no one of the 6 cases here presented was there a central scotoma. A Foster Kennedy syndrome was present in 1 and a paradoxic syndrome was present in another. A feature of interest, and I believe of some importance from a diagnostic standpoint, is the rapid deterioration of vision, once the patient has complained of blurring. The presence of a Foster Kennedy syndrome is an important sign in the diagnosis of a tumor of the frontal lobe; but when it is not present, a progressive concentric contraction, choked disks and an enlarging blindspot on one side should make one think of such a tumor.

GROUP 2: TUMORS IN THE REGION OF THE CHIASM AND THE MIDBRAIN

CASE 7.—J. S., a man aged 42, was admitted to the Brooklyn Hospital on March 24, 1934, with a history of daily headaches and failing vision in the left eye since May 1933. The right eye had been crossed since childhood and had never had vision. The first symptom was a sudden blurring of vision before the left eye, so that the patient ran his automobile into a trolley car. Three months later he was admitted to a hospital in New York. A summary of the report made at this hospital follows:

The patient was admitted to the hospital on Oct. 18, 1933, with a history of blurred vision for the past three months. He had been a heavy drinker of alcohol and beer. A week before admittance he had twitching movements of his eyes, with pain. There was no loss of consciousness or vomiting. There were a right convergent strabismus and loss of sight. The left nerve head was pale. There was tenderness to pressure along the nerves of both lower extremities. The patient was disoriented; there were confabulation and memory defects. The visual fields were good, and the blindspots were not enlarged. A diagnosis was made of alcoholic intoxication with peripheral and optic neuritis.

Ocular examination on April 12, 1934, showed vision of 5/200 in each eye. The pupils were equal and regular and reacted to light and in accommodation. There was a right convergent strabismus. There was complete loss of color perception in the left eye, but color perception in the right eye, the convergent one, was perfect. Examination with a 2 mm. white test object showed almost complete loss of the nasal field in the left eye, with sparing of the macula and in addition some concentric contraction of the temporal field. The right eye showed a loss of about one half of the temporal field, with some contraction of the nasal field.

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9. Kennedy, F.: Retrobulbar Neuritis as an Exact Diagnostic Sign of Certain Tumors and Abscesses in the Frontal Lobes, Am. J. M. Sc. **142**:355, 1911.

10. Lillie, W. I.: Ocular Phenomena Produced by Basal Lesions of the Frontal Lobe, J. A. M. A. **89**:2099 (Dec. 17) 1927.

In view of the results of the ocular examination, it was thought that the patient had a suprachiasmal tumor which pressed on the left optic nerve tract.

Laboratory examinations gave negative results. Roentgenographic examination of the skull and optic canals also gave negative results. Several studies of the visual fields, the last on May 3, showed substantially the same findings. Notes made by Dr. E. J. Browder on May 1 contained the following data: There was apparently an organic lesion. The character of the defects of the visual fields pointed to the parasellar zone on the left side. The condition was most probably a meningioma of the left sphenoid ridge just lateral to the tuberculum sellae.

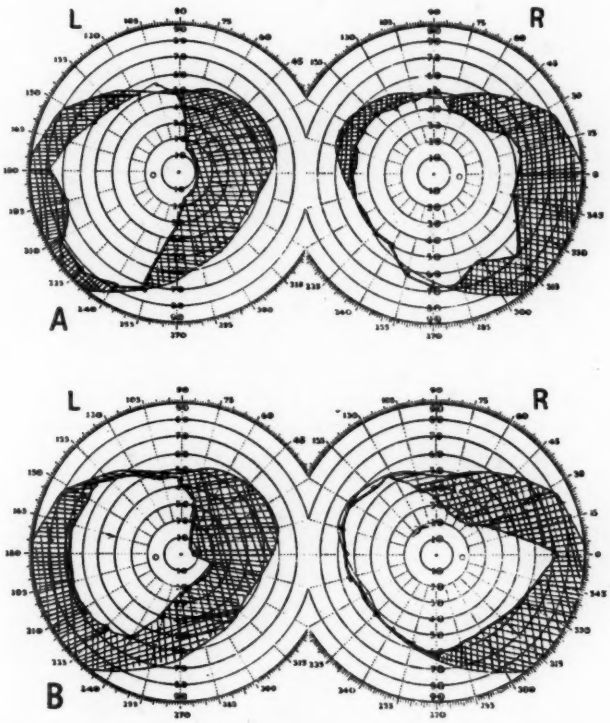

Fig. 7 (case 7).—*A*, visual fields of J. S., taken on April 12, 1934, with a 2 mm. white test object at 250 mm. Vision was 5/200 in each eye. *B*, visual fields of the same patient, taken on May 3, with a 2 mm. white test object at 250 mm. Vision was 5/200 in each eye.

On May 8 Dr. Browder turned down a bone flap in the left frontal region and exposed the sphenoid arch. This was followed medially where the tumor was expected to be, but none was found. When the left optic nerve was visualized, however, he could see a bulbous swelling of the nerve trunk just before it entered the left optic foramen. It was clear to him that the tumor was located within the nerve itself. Several fair-sized blood vessels could be seen coursing over the upper surface of this tumor. An incision was made into the mesial part of the tumor, and several small fragments were taken for histologic study. Dr. Browder decided to get the histologic report before further resection was done, because it would be necessary to sacrifice a portion of the chiasm. As the histologic

study of the fragments revealed them to be edematous nerve tissue with no suggestion of tumor tissue present, nothing further was done.

When the patient was last seen on Sept. 30, 1934, he was completely blind. In spite of the negative histologic report, Dr. Browder felt that we were dealing with a tumor of the optic nerve of unknown origin. The defect in the temporal portion of the right visual field pointed to an extension upward of the tissue into the chiasm involving the crossed fibers of the right optic nerve. Against an inflammatory cause and in favor of a tumor was the absence of atrophy of the optic nerve in the right eye, with the presence of good color perception, which was absent in the left eye.

Case 8.—M. W., a girl aged 17, was seen on Oct. 3, 1931, with a history that about two years before she had lost considerable weight. About a year later a fever developed. Her condition was diagnosed as glomerulonephritis, with uremia. At that time she had headache, nausea and vomiting. These symptoms cleared, and soon after she began to gain a good deal of weight, and her menses

Fig. 8.—Drawing of operative findings by Dr. E. J. Browder.

became scanty and irregular. She also began to become dull mentally. For the past several months she had been under the care of a "gland specialist," who had been giving her injections. She had no headaches, blurring of vision or diplopia.

Vision was 20/20 in each eye. The margins of both disks were somewhat blurred, and the temporal portions were slightly pale. The blood vessels were normal. There was some loss of both temporal fields, particularly of the lower part of the right field, and both blindspots were somewhat enlarged.

A diagnosis of suprasellar cyst was made, and the patient was asked to return in a month for further study. She returned two months later, December 5, saying that she was feeling stronger and more ambitious, but that she was still gaining weight and that her menses were still scanty.

Vision was 20/20— in the right eye and 20/100— in the left eye. The right disk showed slight pallor in the temporal portion. The margins of the left disk were definitely edematous, though not measurable. The pallor in the temporal portion was more marked. Examination of the visual fields showed a cecocentral scotoma and a marked defect in the lower part of the left temporal field. The right visual field showed little change.

The patient went to a prominent neurologist in New York, who in addition elicited a history of diabetes insipidus. A flat roentgenogram showed definitely that there was no tumor of the pituitary gland. The sella turcica appeared half the normal size for a girl that age and seemed flattened from above.

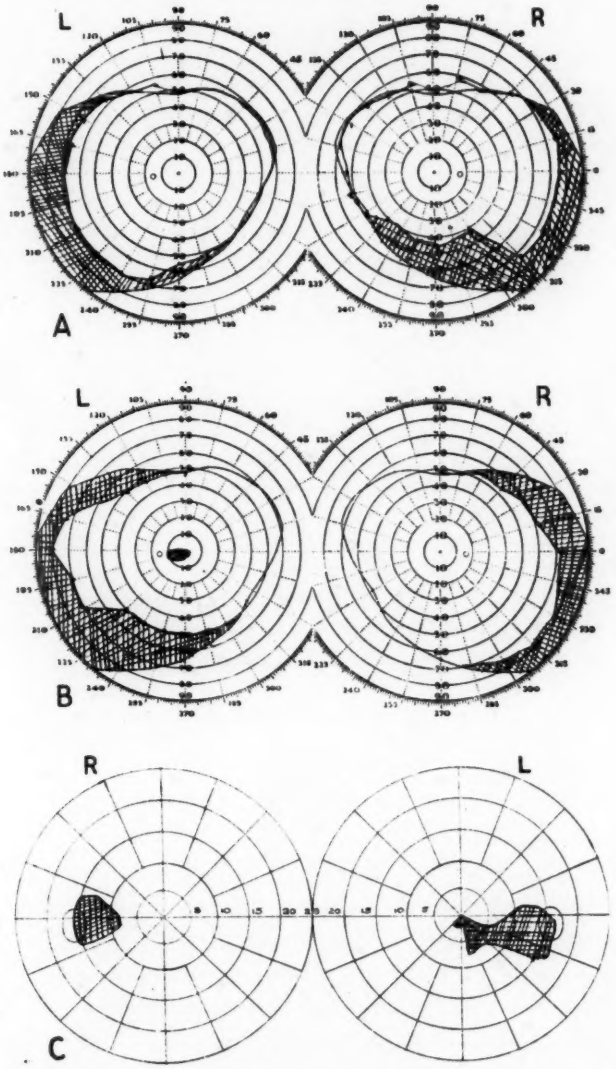

Fig. 9 (case 8).—*A*, visual fields of M. W., taken on Oct. 3, 1931, with a 1 mm. white test object at 250 mm. *B*, visual fields of the same patient, taken on December 5, with a 1 mm. white test object at 250 mm. *C*, visual fields, taken on December 5, with a 2 mm. white test object at 1,000 mm.

It was the neurologist's impression that the patient might have a suprasellar cyst but that the weight of evidence was against such a diagnosis. As he stated his impression, "Her amenorrhea, her obesity and diabetes insipidus could be accounted

for by a lesion on the floor of the third ventricle in approximation of the infundibulum." He referred the patient to an ophthalmologist, who found the same defects in the visual fields previously described; but it was his opinion that because the defect was ventral the lesion was not on top but under the nerve.

This interpretation of the visual fields is not in accord with the present conception of the pathways of the nerve fibers from the retina to the brain. Henschen [11] and others have conclusively shown that the nerve fibers from the retina, with the exception of the macular fibers, keep the same relative position from beginning to end, i. e., fibers from the upper part of the retina, which supply vision in the lower visual fields, are situated in the upper part of the nerve tract and radiation, so that pressure from above will cause loss of the lower visual fields and pressure from the temporal side will cause loss of nasal fields.

However, the neurologist and the ophthalmologist referred the patient to a neurosurgeon, expressing their uncertainty as to the diagnosis. He agreed with them, and referred her back for further observation. She was observed monthly until her vision was completely lost in both eyes, when she went to the Johns Hopkins Hospital, where she was operated on, July 12, 1932.

The following report was taken from Dr. Dandy's notes: When the left lateral ventricle was tapped, fluid spurted out under high pressure. About 20 cc. of fluid was removed, and an equal amount of air was injected. The right lateral ventricle did not fill. The third ventricle filled only in part and showed a filling defect due to a tumor. There was no dislocation of the lateral ventricles. The patient was kept in the hospital for two weeks because of a fever, the temperature ranging from 102 to 103.5 F. The leukocyte count was around 10,000. No infection was found to account for the fever, and it was finally concluded that it was central in origin. The patient had been extremely drowsy throughout her stay in the hospital, sleeping most of the time, but she could be roused. Pressure could not account for her drowsiness, because the ventricular dilatation was too slight, so it had to be explained solely on the basis of the part of the brain involved by the infiltrating lesion.

A preoperative diagnosis of an infiltrating lesion of the floor of the third ventricle was made.

Operation revealed the third ventricle to be full of gliomatous tumor tissue. The tumor tissue, being on the floor of this ventricle, had directly involved the visual fibers.

In view of the course of the disease, it is interesting to speculate on the cause of the fever in 1929. Did the patient have glomerulonephritis, of which she gave no later symptoms or objective findings, or did she have hyperthermia of central origin with diabetes insipidus?

Also of interest are the marked symptoms of pituitary dysfunction, in spite of the fact that roentgenograms showed no direct pressure on the pituitary gland by the absence of an enlarged sella turcica.

CASE 9.—C. A., a man aged 27, was seen on March 8, 1937, with a history that about twelve years before he noticed a beginning loss of libido. He went to an internist, who treated him with glandular preparations. Shortly after, he complained of visual disturbances, and glasses were prescribed, with relief from his

11. (*a*) Henschen, S. E.: Zur Anatomie der Sehbahn und des Sehzentrums, Arch. f. Ophth. **117**:403, 1926. (*b*) Rönne, H.: Pathologisch-anatomische Untersuchungen über alkoholische Intoxikationsamblyopie, ibid. **77**:1, 1910.

symptoms. For some time he continued to receive glandular therapy. Every now and then he would have a severe headache. Vision began to deteriorate, but so slowly that he was not conscious of its loss until it was well advanced In the meantime he had changed doctors several times but continued to receive glandular treatment.

In November 1936, while in Chicago, his headaches became severe and persistent and were accompanied by projectile vomiting. He went to an ophthalmologist, who referred him to Dr. Loyal Davis. Studies of the visual fields showed a defect in the upper temporal portion of the right field and a tendency toward a concentric contraction in the left field.

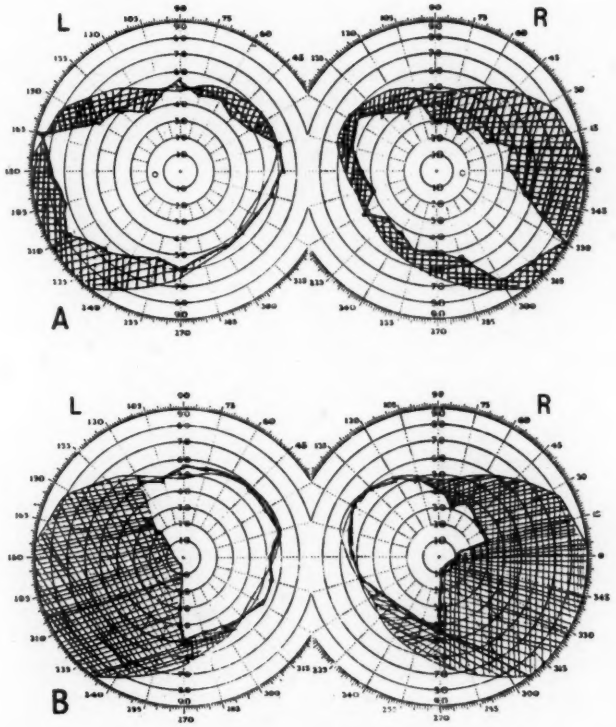

Fig. 10 (case 9).—*A*, visual fields of C. A., taken on Nov. 10, 1936, with a 4 mm. white test object. *B*, visual fields of the same patient, taken on April 20, 1937, with a 2 mm. white test object at 330 mm. Vision was 20/70 in each eye.

On November 16 Dr. Loyal Davis performed a right transfrontal osteoplastic craniotomy and found a cyst extending anteriorly to the chiasm rather than posteriorly. When it was opened a large amount of yellow fluid containing fatty deposits was evacuated. Immediately below the chiasm and pressing upward into its structure was a large globular irregular mass of cholesterol. It extended into the tissue of the chiasm. Part of the wall of the cyst and as much of the cholesterol as possible were removed without severing the chiasm. The chiasm was so involved by this mass that Dr. Davis was certain the visual fields obtained were absolutely incorrect (obtained by clinical clerks). The damage was in the center, and one would expect the visual fields to show bitemporal hemianopia.

After operation the patient's vision improved. Physical examination showed the patient to be short and slight of build, with no hair on the face. He had small delicate hands and feet with the voice of a preadolescent boy. He had complete loss of sexual power.

Ocular examination on April 20, 1937, showed vision of 20/70 in each eye, which improved with correction. Both disks showed advanced primary atrophy. The visual fields showed practically complete bitemporal hemianopia, which corresponded to what Dr. Davis thought should have been present at the first examination.

Case 10.—L. T., a woman aged 50, entered the Brooklyn Hospital on Oct. 5, 1936, with a history that thirteen years before she had noticed a tingling of her hands and feet. One year later she began to grow larger in all dimensions until four years before admission to the hospital. During this period she gained about 100 pounds (45 Kg.), and her hands and feet grew so large that she had to wear men's sizes. Two years before admission she began to have pains in her legs. One and a half years before a tonsillectomy was performed in a

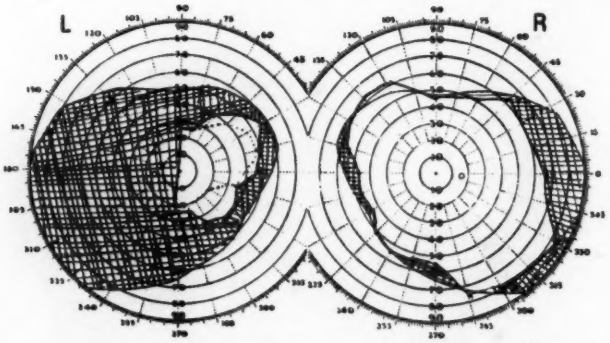

Fig. 11 (case 10).—Visual fields of L. T., taken on Oct. 8, 1936. Vision in the left eye was limited to perception of fingers at 1 foot. The visual field with a 15 mm. white test object at 250 mm. is indicated by the dotted line; that with a 40 mm. white test object is indicated by the heavy black line. The right eye was tested with a 2 mm. white test object at 250 mm. Vision was 13/13.

hospital in Brooklyn. About a year before she began to have polyuria and polydipsia. About this time she received roentgen treatment to the region of the pituitary gland, which was followed by loss of vision in the left eye. Her periods started at the age of 13 and were normal until her menopause at 44.

Physical examination revealed a person of the typical acromegalic type. Dr. Browder made a diagnosis of acidophilic adenoma.

Ocular examination on October 8 showed vision in the right eye of 13/13; vision in the left eye was limited to perception of fingers at 1 foot (30 cm.). The left pupil was slightly larger than the right, but both reacted to light and in accomodation. The margins of the right disk were blurred, although no measurable edema was present. There was no pallor of the temporal side. The veins were not unusually full, and there were no hemorrhages or exudates. The margins of the left disk were blurred. The entire disk was pale, the temporal side being more so. With a 2 mm. test object there was a tendency toward a concentric contraction of the right visual field, with a greater loss in the upper

temporal portion. The smallest test object that could be used for the left eye was a 40 mm. target. With this test object there was complete loss of the temporal portion of the field, with a marked contraction of the nasal side.

On the basis of the results of the ocular examination, it was thought that a tumor of the pituitary gland was pressing on the left optic nerve, with beginning involvement of the right crossed fibers.

On October 19 Dr. Browder, through a transfrontal approach, removed an acidophilic adenoma, a knuckle of which had broken through the dorsum sellae.

CASE 11.—D. F., a man aged 42, came to the dispensary of the Brooklyn Hospital on Sept. 26, 1934, with a history that a year previously a herniorrhaphy had been performed (right inguinal) with spinal anesthesia. Since then he had severe headaches in the frontal and parietal regions. Five months ago before coming to the dispensary he noticed that his right upper lid drooped and that vision in the right eye was lost. He had never vomited.

Ocular examination on September 27 showed ptosis of the right upper lid which, while the patient was under observation, varied from slight to complete.

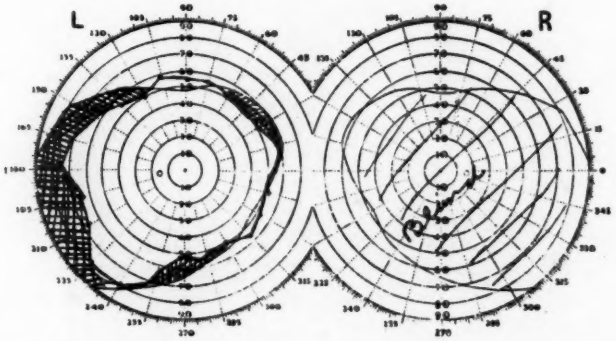

Fig. 12 (case 11).—Visual fields of D. F., taken with a 2 mm. white test object at 250 mm.

The eye was practically fixed in the midline, having but slight movement in any direction. The right pupil was widely dilated and reacted to consensual but not to direct light. The left pupil reacted well both to direct and to consensual light. Vision in the right eye was limited to uncertain perception of light and that in the left eye equaled 20/20. The right disk showed moderate primary atrophy. The left visual field was normal. There was tenderness on percussion over the right orbit.

On the basis of the results of the ocular examination, it was thought that there was a tumor in the apex of the right orbit, the only point against this diagnosis being the absence of exophthalmos.

The roentgenographic report was negative, except for mention that the posterior clinoid processes were not visible.

The patient was admitted to the hospital. On October 5 Dr. Browder made the following observations: The patient vomited for the first time the previous day. There was pain over the right side of the head. Nothing, however, was heard on auscultation. The eye was held in external rotation (this was evidence of the varying state of the ocular paralysis). There was no loss of sense of smell.

It was thought that the condition was either a tumor in the apex of the orbit or one involving the sphenoid fissure. The possibility of it being a meningioma of the right tuberculum sellae or of the medial aspect of the right sphenoid ridge was recognized. However, the first impression, in view of the normal left visual field, was that the condition was a metastatic lesion in the situations previously mentioned. Exploration was advised.

Ocular examination on October 11 showed no change over the previous findings, except that the atrophy of the right disk was more pronounced.

Another roentgenogram made on October 8 was normal.

On October 13 neurologic examination revealed the following symptoms: (1) a diminution of pain and temperature sense on the right, (2) a diminution of tactile discrimination and vibratory sensation on the right, (3) normal stereognosis and (4) increased spinal fluid pressure.

The patient refused to have an operation performed and was discharged from the hospital.

On November 13 he was admitted to the Neurological Institute New York, when the following findings were recorded: After the patient's discharge from the Brooklyn Hospital vision in the left eye began to fail and became progressively worse. The deep reflexes on the right were hyperactive, and the Babinski sign on the right was positive. There was marked atrophy of the optic nerve on the right, and the left visual field was greatly restricted. The serologic reactions and the chemical composition of the blood were normal.

The first roentgenographic report stated that the condition was an intracranial expanding lesion and that aneurysm was to be considered, but definitely not a tumor of the pituitary gland.

A second roentgenogram strongly suggested that an aneurysm of the right internal carotid artery or of the posterior communicating artery was present.

According to the encephalographic report an expanding lesion, slightly posterior to the chiasm was present, which was probably a tumor, an adenoma of the pituitary gland, and not an aneurysm.

On December 1 a right transfrontal craniotomy exposed a tumor in the suprasellar region, which was partially removed. The pathologic diagnosis was that of chordoma.

The postoperative diagnosis was chordoma of the middle fossa.

CASE 12.—W. C., a Negress aged 32, came to the neurologic clinic of the New York Post-Graduate Medical School and Hospital on Oct. 16, 1937, with a history that since December 1936 her vision had been failing. In April 1937 her menses stopped, and she began to have occasional frontal headaches. Occasionally she vomited, especially in the morning. She had continuous thirst and an excessive craving for sweets. She had been going to doctors and clinics, but her vision rapidly became worse.

Neurologic examination disclosed a well developed Negress without any physical signs of a deficiency in the pituitary secretion, such as adiposity or dull mentality. The fundi were normal, and neurologic examination gave negative results.

The opinion was that the condition was probably a suprasellar cyst.

Ocular examination on October 22 showed vision in the right eye to be limited to perception of hand movements on the nasal side. Vision in the left eye was limited to perception of fingers at 2 feet (60 cm.). The visual fields were unreliable but suggested left homonymous hemianopia. The pupils were equal and regular and reacted slowly to light and well in accommodation. The fundi were normal.

In view of the normal fundi and the history of such long-standing blindness, a lesion of the posterior cortex was suggested.

A week later the patient was again examined in the ophthalmic clinic by another observer, and at this time she was completely blind and the fundi were still normal.

Flat roentgenograms on October 23 were completely normal.

A month later the patient went to the Neurological Institute of New York, where Dr. John Scarf found the right eye to be completely blind and the other eye to have a temporal hemianopia to strong light.

At this time the disks showed evidence of primary atrophy. The plain roentgenograms were normal. An encephalogram revealed evidence of a suprasellar cyst.

On November 27 a large suprasellar cyst was exposed, and about 30 cc. of xanthrochromic fluid was evacuated. The following day the patient could count fingers with either eye at 5 feet (152 cm.).

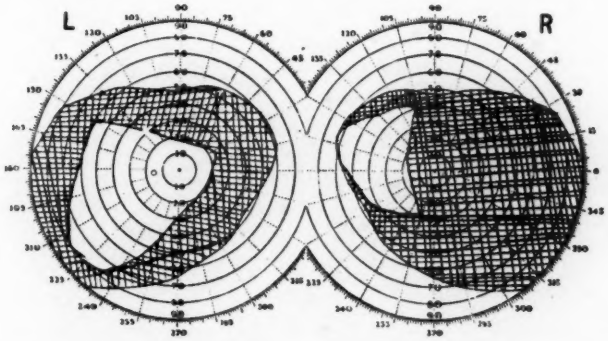

Fig. 13 (case 12).—Visual fields of W. C., taken on Oct. 22, 1937. Vision in the right eye was limited to perception of hand movements on the nasal side; that in the left eye was limited to perception of fingers at 2 feet.

A great deal of the loss of vision must be explained on a hysterical basis. Though the patient was so blind that she had to be led around, the fundi remained normal for a while and even later showed only early atrophy of the optic nerve. After operation, Dr. Scarf expressed astonishment that the next day the patient was able to count fingers at 5 feet with either eye.

Case 13.—J. G., a Negro aged 34, was admitted to the Neurological Institute of the Brooklyn Hospital on Feb. 24, 1938, with a history that until 1929 he was of the average normal height of the rest of his family. At this time he wore size 9 shoes. Then he began to grow in size; his features began to change, and his hands and feet began to grow progressively larger. Until 1933 there were no other symptoms. At this time vision in the right eye began to fail; the hair on the face and pubis became more scant, and his sexual power was impaired. He also became lethargic, so that in time he was unable to do any work. At the time of admission to the hospital he wore size 12½ shoes, and there was complete loss of sexual power. He was also several inches taller than in 1929.

Examination showed a very tall Negro, who appeared ill and toxic. He had the typical features of a person with acromegalia. Mentally, he was dull.

A diagnosis of eosinophilic adenoma of the pituitary gland was made. A flat roentgenogram taken on March 9 showed the bones of the skull to be rather thin and the sella turcica to be immense. The anterior and posterior clinoid processes were partially eroded.

The impression on the basis of the roentgenographic picture was that the condition was a tumor of the pituitary gland.

Ocular examination on March 16 showed advanced atrophy of the right disk. There was imperfect perception of light. The left disk was of good color. There was loss of the temporal field, with sparing of the macula.

A diagnosis of tumor in the region of the sella turcica was made.

On March 18 Dr. E. J. Browder removed a large cystic degenerated tumor of the pituitary gland through a flap in the right frontal region.

Case 14.—A. D., a woman aged 29, came to the neurologic clinic of the New York Post-Graduate Medical School and Hospital on Nov. 17, 1937, with a

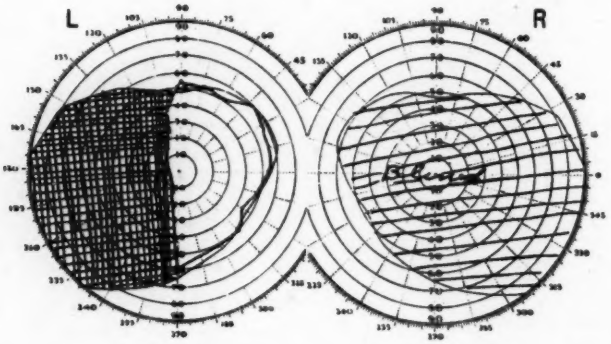

Fig. 14 (case 13).—Visual fields of J. G., taken on March 16, 1938, that for the left eye being plotted for a 2 mm. test object at 330 mm.

history that since July 1937 her vision had been getting progressively worse. For the past five months she had been having periodic headaches which arose from the frontal region and radiated to the back of the head. They usually came about once a month and lasted from one to two days. The headaches were severe. She also vomited frequently. Her menstrual periods stopped about seven years previously, and she had not had one since. She had not lost or gained any weight, and there was no history of polyuria or polydipsia. At 9 years of age a nail pierced the right eye, and since then she had never been able to see with that eye. An ophthalmologist had made a diagnosis of bilateral central scotoma.

Flat roentgenograms taken on Jan. 11, 1938, showed the following picture: The sella turcica was much distended. The posterior clinoid processes were hazy, and the sphenoid sinuses were encroached on. All these findings were thought to point to the presence of an expanding neoplasm in the region of the sella turcica.

Ocular examination on January 12 showed vision in the right eye to be limited to perception of hand movements. It was not improved with correction. There was a scar in the upper right quadrant of the cornea. The pupil was drawn to

the scar by an adherent iris. The disk showed moderate primary atrophy. Vision in the left eye was 5/200; it was not improved with correction. The disk showed moderate primary atrophy. Examination of the visual fields revealed a bitemporal macular defect and a defect in the lower temporal quadrant of the left eye. The macular defect in the right eye involved the entire central area. This, I felt, was caused by the long-standing amblyopia.

It was thought that the bitemporal macular defects were due to pressure on the crossed macular tracts in the chiasm and that the defect in the inferior quadrant indicated pressure from above.

A diagnosis of a suprasellar cyst was made.

On January 28 Dr. Mac Lean turned down an osteoplastic flap in the right temporofrontal region. In the region of the sella turcica there was a cyst which encroached on the chiasm posteriorly and superiorly. The cyst was needled, and 8 cc. of thick yellow brown fluid was aspirated. The cyst was then incised, and tumor tissue was curretted out.

A postoperative diagnosis of tumor of the pituitary gland with cystic degeneration was made.

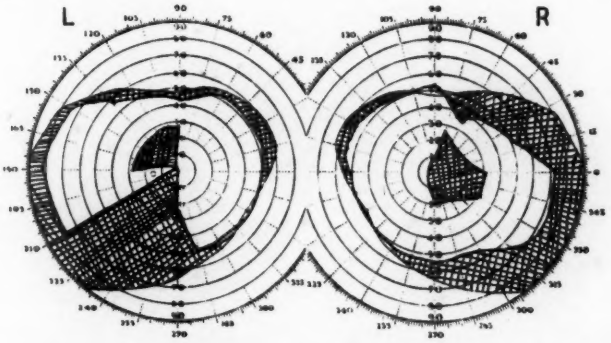

Fig. 15 (case 14).—Visual fields of A. D., taken on Jan. 12, 1938, with a 5 mm. white test object at 250 mm. Vision was 5/200 in the left eye; that in the right eye was limited to perception of hand movements.

The second group of cases can only emphasize what has so often been emphasized by Cushing, Walker and others,[12] that lesions in the region of the chiasm, owing to the close proximity of so many structures, can give rise to puzzling changes in the visual fields as well as to unusual physical signs.

It hardly seems possible that persons with such outstanding signs of acromegaly as those considered in cases 10 and 13 would be allowed to go for so many years without diagnosis and therapy. The patient in

12. (*a*) Walker, C. B., and Cushing, H.: Chiasmal Lesions with Especial Reference to Homonymous Hemianopsia with Hypophyseal Tumor, Arch. Ophth. **47**:119, 1918. (*b*) Horrax, G., and Putnam, T. J.: Distortions of the Visual Fields in Cases of Brain Tumor, Brain **55**:499, 1932. (*c*) Cushing, H., and Heuer, G. J.: The Distortions of the Visual Fields in Cases of Brain Tumor, Bull. Johns Hopkins Hosp. **22**:190, 1911.

case 9 should have been operated on long before such complete loss of pituitary function developed; nor should the patient in case 12 have been allowed to become completely blind.

Of great interest is case 8, in which, in the presence of a tumor of the third ventricle, there were all the symptoms, physical and endocrinologic, as well as changes in the visual fields, suggestive of a suprasellar cyst. Outside of the hyperthermia, there were none of the symptoms usually associated with tumor of the third ventricle, such as loss of upward gaze, altitudinal anopsia and high grade papilledema.

In no other group of cases can studies of the visual fields, and especially follow-up studies, be so fruitful of good results. Any patient showing evidence of pituitary dysfunction and changes in the visual fields which can be attributed to a lesion in the region of the chiasm and

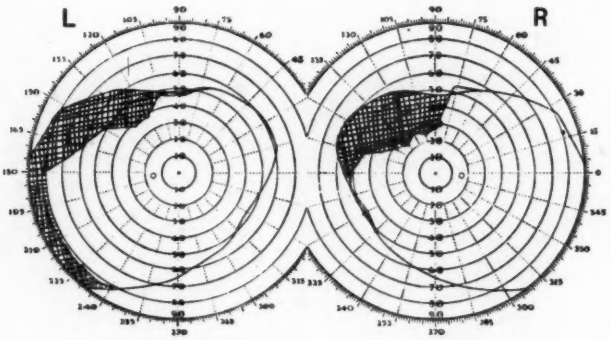

Fig. 16 (case 15).—Visual fields of I. S., taken on Oct. 13, 1929, with a 1 mm. white test object. Vision was 20/15 in each eye.

progressive loss of vision is entitled to an exploratory operation instead of waiting for blindness to supervene.

GROUP 3: TUMORS POSTERIOR TO THE CHIASM

CASE 15.—I. S., a man aged 26, was seen on Oct. 11, 1929, with a complaint of double vision. For the past year he had been having occasional attacks of unconsciousness. These attacks lasted for a few seconds and were accompanied by either visual or auditory aura. The auditory aura consisted of beautiful angelic voices, while the visual aura were made up of angles or corners of rooms, never of persons. The aura were always the same. He never had hallucinations of taste or smell, nor did he ever have convulsions. About one year before the present examination, at the commencement of his complaint, the patient had profuse rhinorrhea and a severe headache and vomiting. His condition had been diagnosed as petit mal several times.

On September 13 he saw double for the first time. The diplopia persisted. In October 1929 he again had a severe attack of rhinorrhea and headache but no vomiting. The attack lasted ten minutes and was accompanied by blurred vision. Physical and neurologic examination gave completely negative results.

Ocular examination showed vision of 20/15 in each eye. There was no ocular palsy and no nystagmus. There was a slight, barely perceptible convergence of the right eye when the patient looked at a distance. The pupils were equal and regular and reacted to light and in accommodation. Examination of the fundi showed about 1.5 D. of swelling of the right disk and about 2.5 D. of swelling of the left disk. There were no hemorrhages or exudates. The patient had homonymous diplopia, which increased for distance and disappeared at the reading distance. The diplopia did not increase to the right or left, a true divergence paralysis.

Perimetric study revealed hemianopia in the upper left quadrant, and a diagnosis of a tumor deep in the right temporal lobe was made.

On November 5 Dr. Dandy removed a large deep-seated glioma, and with it practically the entire right temporal lobe. The first symptom to disappear was the diplopia. Dr. Dandy stated in a letter that the only localizing sign the patient ever had was the defect in the visual field.

CASE 16.—C. F., a man aged 39, entered the Brooklyn Hospital on March 5, 1935, with a complaint of severe headaches for the past year. Two weeks before

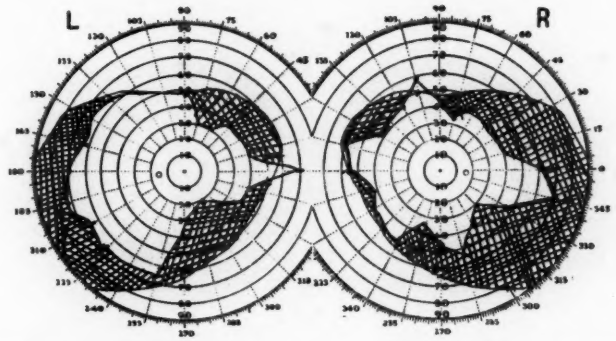

Fig. 17 (case 16).—Visual fields of C. F., taken on March 6, 1935, with a 2 mm. white test object at 250 mm.

admission the headaches became more severe, and he vomited a great deal. On the day of admission he became stuporous. Examination of the fundi showed the right disk to be swollen about 2.5 D. and the left, about 1.5 D. The disks showed evidences of secondary atrophy. There were a few hemorrhages around the disks. The next day the patient was fairly alert, irritable and uncooperative, but satisfactory visual fields were obtained. These showed an irregular concentric contraction and right hemianopia, the lower portions of the fields being particularly involved.

A diagnosis of tumor of the left frontoparietal region was made. On March 7 examination by a neurologist revealed the following data: The patient was clear mentally and was complaining of severe frontal headaches, mostly behind the eyes. His mental processes were slow, but not strikingly so. He had noticed a queer numbish feeling in the right upper extremity for the past week, and there was a drifting of the right upper extremity. There were no gross stereognostic changes. There was bilateral ankle clonus but no Babinski sign.

In view of these findings, it was thought that the patient had a tumor of the inferior parietal lobe.

On March 10 Dr. Browder turned down a bone flap in the left parietal region (somewhat posterior), and a soft tumor in the lower parieto-occipital region was scooped out. It proved to be a spongioblastoma.

Case 17.—J. M., a man aged 23, came to the dispensary of the Brooklyn Eye and Ear Hospital on Sept. 2, 1932, with a history of headaches for the past year and diplopia for the past week. The headaches came on about two or three times a week. A month previously he noticed numbness and weakness of his left side, and he began to vomit. He entered a local hospital, where he was kept for ten days and was discharged without a diagnosis. A week before he came to the dispensary diplopia developed.

Ocular examination showed vision of 20/25— in each eye. The right eye was slightly turned in. There was homonymous diplopia, which increased for distance and disappeared at the reading distance. The diplopia did not increase to the right or left, a true divergence paralysis. The right disk showed about 2.5 D. of swelling and the left, somewhat less. The visual fields showed hemianopia in the upper left quadrants. A roentgenogram of the skull on October

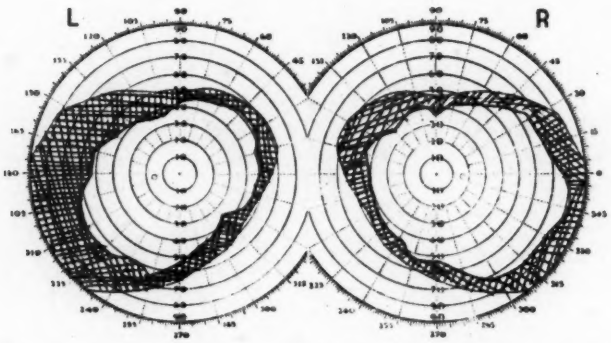

Fig. 18 (case 17).—Visual fields of J. M., taken on Sept. 5, 1932, with a 2 mm. white test object at 250 mm.

6, at the Brooklyn Eye and Ear Hospital, did not show any evidence of an intracranial abnormality.

A diagnosis of tumor of the right temporal lobe was made.

The patient was admitted to Dr. Browder's service at the Long Island College Hospital on October 14. Physical examination revealed choking of the disks and active knee and ankle jerks but no localizing signs. Perimetric studies at this hospital showed general contraction of the visual fields, which was more noticeable on the left. Ventriculographic examination by Dr. Mendelson showed tumor of the brain, probably of the right frontal lobe.

On October 16 a bone flap was turned down on the right side, and a tumor of the right ventricle was exposed. It arose from the medial wall about opposite the junction of the posterior and the temporal horn of the right ventricle. It was fairly well circumscribed and attached to the choroid plexus.

A diagnosis of ependymoma of the right ventricle was made.

Case 18.—M. B., a woman aged 46, entered the Brooklyn Hospital on Sept. 23, 1933, with a history that three weeks previously severe pains suddenly developed across the top of the head. Later that day the pains became localized to the

right frontal area. Since then the headaches had been constant and knifelike. Vomiting began simultaneously with the headaches and was so severe that she could not retain even water. She remained in bed, because even raising her head made her dizzy. A local physician diagnosed the condition as sinusitis, and much medication was prescribed. From November 1932 to June 1933 she had frequent nose bleeds, but none since then. The house surgeon at the hospital made the following observations: Vision was poor. There was a suggestion with a gross test of left homonymous hemianopia. There was bilateral ptosis, more on the right than on the left. The right pupil was smaller than the left and both reacted sluggishly to light and in accommodation. There were bilateral facial paresis and a slight drift of the right arm. There was bilateral papilledema.

On the basis of the results of the ocular examination, it was thought that the condition was an abscess of the right hemisphere of the brain or a tumor of the brain involving the midbrain and hypothalamus.

The same day neurologic examination showed the following symptoms: (1) bilateral ptosis, greater on the right than on the left; (2) a defect of upward gaze, more marked on the right; (3) vertical nystagmus when the patient

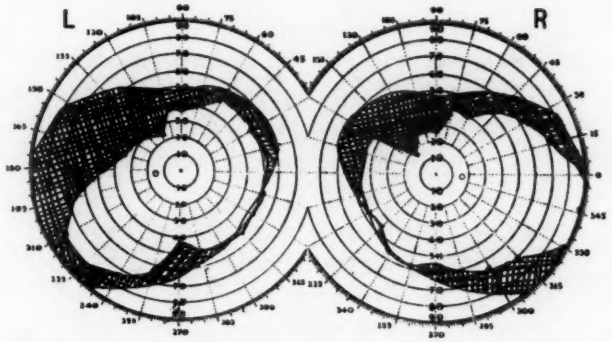

Fig. 19 (case 18).—Visual fields of M. B., taken on Jan. 9, 1934, with a 2 mm. white test object at 250 mm.

attempted to look up; (4) early papilledema, more marked on the right, and (5) exquisite tenderness of the right frontoparietal region to tapping and deep pressure.

The state of the reflexes was not considered of localizing value. Syphilis, because of involvement of the tegmental plate, was ruled out as were also tumor of the brain and encephalitis.

On September 27 Dr. Browder made the following observations: Because of the acute onset and rapid progression of symptoms to a marked state of stupor within a period of three weeks, the amount of choked disks (which had increased tremendously), the absence of upward gaze, the bilateral ptosis and the stiff neck, the condition was probably a posterointraventricular tumor or a lesion of the quadrigeminal plate.

On October 1 a ventriculogram showed a shift to the left, indicating a tumor in the right frontotemporal area.

That day Dr. E. J. Browder turned down a bone flap on the right side, and a tumor 5 cm. in diameter was outlined in the posterior part of the temporal convolution about 1.25 cm. under the cortex.

The pathologic diagnosis was glioblastoma multiforma.

The patient was readmitted to the hospital on Jan. 6, 1934, because of recurrent headaches and vomiting. She was referred for studies of the visual fields, which revealed left homonymous hemianopia. On January 17 the bone flap was reelevated by Dr. Browder, and a recurrent tumor occupying the region of the temporal fossa was identified. The entire temporal lobe of the brain with the tumor was removed.

I did not examine this patient until before the second operation, but the report of the case is included because of its many points of interest. Although all neurologic signs pointed toward a tumor of the midbrain, even the superficial examination of the visual fields made by the house surgeon revealed a left homonymous hemianopia. If this patient had been properly examined by the perimeter, particularly before the vision became too poor, a more exact diagnosis would probably have been made by the neurologists. This case emphasizes again the importance of early and accurate examinations of the visual fields.

In contrast to case 8, the classic signs of a tumor of the third ventricle were present in case 18, but the tumor was in the posterior part of the temporal lobe. The left homonymous hemianopia was the only correct localizing sign, but in the presence of neurologic signs which seemed so definite, little value was placed on it. This can only reemphasize the value of early and correctly plotted visual fields.

Perhaps the loss of upward gaze must be revaluated as a symptom of tumors of the third ventricle. Of 7 cases of tumor of the third ventricle reported by Globus,[13] loss of upward gaze was present in only 1. It seems to be more constantly a sign of tumors of the pineal body, which as a rule give rise to higher intracranial pressure. It may therefore be a sign of remote pressure effects, possibly a generalized pressure on the posterior longitudinal fasciculus.

CASE 19.—E. A., a woman aged 50, was admitted to the Brooklyn Hospital on Feb. 4, 1933. She had never been ill until two months before, when she was awakened at night with a severe headache. The headache had been constant since then.

Three weeks before admission to the Brooklyn Hospital she was admitted to a local hospital, where six teeth were removed, with amelioration of the symptoms. A few days later the headaches recurred, more severe than before. For the past one and a half weeks her vision had blurred and the left eye turned in. Vision rapidly become worse, so that she had only preception of light.

On admission to the hospital the intern noted right hemianopia. The next day the neurologist made the following note: "Because of the poor condition of the patient and the poor vision, localization is not possible. In view of the intern's findings, a left temporal lobe tumor is a likely diagnosis."

Ophthalmologic examination on the following day showed the following facts: The patient was uncooperative and restless. She was able to count fingers at 6 inches (15 cm). There was palsy of the left external rectus muscle. Both disks

13. Globus, J. H., and Silverstone, S. M.: The Diagnostic Value of Defects in the Visual Fields and Other Ocular Disturbances Associated with Supratentoreal Tumors of the Brain, Arch. Ophth. **14**:325 (Sept.) 1935.

were swollen about 5 or 6 D., and there were numerous hemorrhages scattered about the fundi. Gross tests of the visual fields, taken under great difficulty, revealed definite left hemianopia. This was confirmed the next day. Fifteen minutes after this examination the patient lapsed into unconsciousness, from which she did not emerge until after the operation.

Solely on the basis of the findings in the visual fields, Dr. Browder on February 7 turned down an osteoplastic flap in the right temporoparietal region and removed a solid tumor from the right inferior parietal area. This proved to be a fibrillary astrocytoma.

CASE 20.—E. H., a woman aged 34, was admitted to the Brooklyn Hospital on Jan. 10, 1938, complaining of attacks of severe pain in the face and head.

Her illness started in March 1937, with what she stated was a "nervous breakdown." This was characterized by severe headaches, so that she had to give up her work as a school teacher. In April 1937 she had a facial paralysis on the left side, from which she recovered. At that time she had mild attacks of pain in both cheeks. Since November these had been constant and more severe, coming on

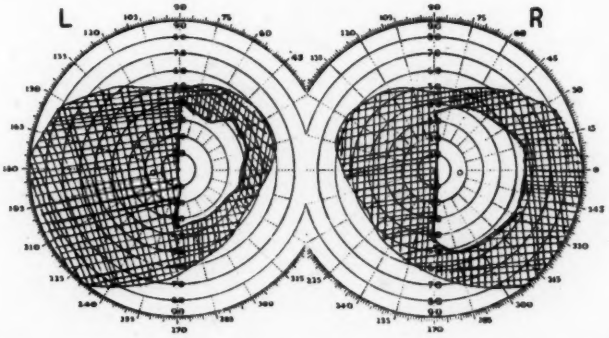

Fig. 20 (case 19).—Visual fields of E. A., taken on Feb. 5, 1933, with the gross finger test.

about a dozen times a day. The pain was sharp. There had been no visual or auditory disturbances. Around December 25 there was a period of vomiting, which lasted for two days. Since the occurrence of the facial paralysis, speech had always been somewhat thick.

In 1936 a physician made a diagnosis of neuritic atrophy of both disks and referred the patient to a hospital in New York, where roentgenograms of the skull were taken. The report, dated May 5, 1936, stated that there were areas of calcification in the left occipital lobe, with moderate accentuation of markings in the lateral frontal area. No interpretation of the findings was made, and nothing further was done.

The attacks of pain in the face were always brought on by cold weather, particularly drafts. The patient was observed during several of these attacks. The facial muscles became rigid, especially those of the lips, which caused the face to become "puckered." The upper lip especially was stiff, and speech became "tongue-tied," or like "baby talk."

Apprehension was impaired, in that the patient was unable to explain the nature of her husband's work, and although a teacher of languages, she was

unable to parse simple verbs. She also found great difficulty in explaining things in detail.

Neurologic examination revealed the following symptoms: (1) paralysis of the right side of the face of the supranuclear type, (2) a peculiar dysarthric speech, (3) slightly hyperactive deep reflexes, (4) completely normal sensory findings and (5) no word blindness of any kind.

A diagnosis of tumor of the left occipital lobe was made.

A flat roentgenogram taken January 11 showed a calcified mass in the left occipital lobe. The sella turcica was enlarged, and the posterior clinoid processes were eroded. There was wavy uneveness of density in the frontal area.

On the basis of the roentgenographic picture, it was thought that the condition was a tumor of the left occipital lobe. Ocular examination on January 14 showed vision of 13/30 + in the right eye and 13/70 + in the left eye. The pupils were equal and regular and reacted to light and in accommodation. There was no nystagmus or ocular palsy. Examination of the fundi showed the disks to be swollen about 3.5 D. There were no hemorrhages or exudates. The visual fields showed right homonymous hemianopia, which in part cut through the maculas.

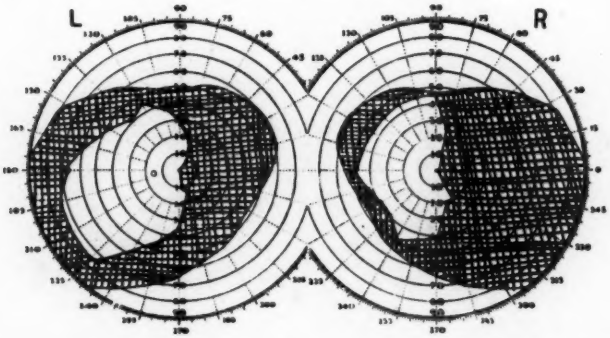

Fig. 21 (case 20).—Visual fields of E. H., taken on Jan. 14, 1938, with a 2 mm. white test object at 330 mm. Vision in the left eye was 13/70 + and the right eye, 13/30 +.

On the basis of the results of the ocular examination, it was thought that the condition was a tumor of the left occipital lobe.

On January 17 Dr. E. J. Browder turned down an osteoplastic flap in the left occipital region. An encapsulated cystic tumor was found, and from 8 to 10 cc. of yellow fluid was removed. The tumor mass was a whitish yellow, similar in color, but of firmer consistency, to the brain. Blunt dissection showed the tumor to have pushed the parietal region in front of it rather than to have invaded it. The entire left occipital lobe was removed.

Pathologic examination proved the growth to be an astrocytoma.

GROUP 4: TUMORS OF POSTERIOR FOSSA

CASE 21.—J. A., a boy aged 14, was seen on Feb. 24, 1938, with a history that about five years previously he had been struck on the head with an oil drum. The parents stated that he was unconscious for a little while but was not taken to a hospital. He was perfectly well until about a year before the present trouble, when he began to complain of headaches. Little attention was paid to his complaints, as

they were not severe. During the previous summer he had fallen and was unconscious for a while. Three months before he had a severe spell of vomiting, which lasted for three weeks. At that time he had a cold with fever. A physician was called, and he found pus and sugar in the urine. Another physician could not corroborate these findings. For the past few months he had been acting peculiarly. He had gained about 17 pounds (1.7 Kg.) in the past two months. Recently he had been having sensations of falling backward while walking.

Ocular examination showed that vision in the right eye was limited to perception of fingers at 2 feet. Vision in the left eye equaled 20/70. There was no nystagmus or ocular palsy. Both pupils were dilated but reacted to light and in accommodation. There was about 6 D. of swelling of the right disk and 5 D. of swelling of the left disk. The arteries were extremely small, and the veins were dilated, but no hemorrhages were visible. Both disks showed evidences of advanced secondary atrophy. The right visual field showed an extreme concentric contraction to a large test object (40 mm. white), while the left showed a concentric contraction to a 10 mm. test object.

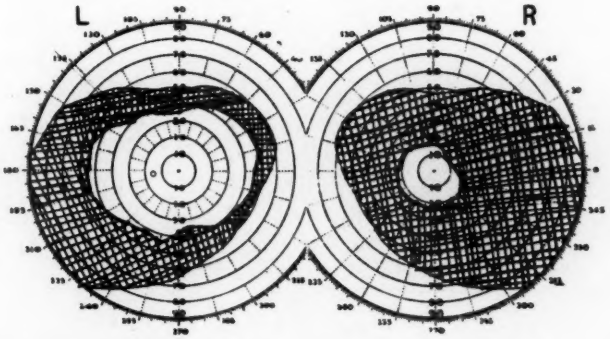

Fig. 22 (case 21).—Visual fields of J. A., taken on Feb. 24, 1938, with a 10 mm. white test object for the left eye and a 40 mm. white test object for the right eye. Vision in the left eye was 20/70; that in the right eye was limited to perception of fingers at 2 feet.

A diagnosis of tumor in the posterior fossa was made, and the patient was referred to the Brooklyn Hospital, where he was admitted on Februray 25. A summary of the neurologic findings follows: Roughly, odors were identified. There was a transient and slight paresis of the right seventh nerve of the supranuclear type. There was a drift of both extremities, but the right tended to drift more. There was a slight terminal ataxia in the right finger to nose test. There was a right Babinski sign, and the patient had a tendency to go to the right on walking. He stood equally poorly on either foot. Sensory examination gave negative results. Examination of the left visual field by the intern showed a loss in the nasal portion. The right visual field was not plotted.

On the basis of the results of the ocular examination, it was thought that the condition was a tumor deep in the left parieto-occipital region. This diagnosis was concurred in by the visiting neurologists.

A flat roentgenogram of the skull taken on February 26 revealed the following picture: The bones of the skull seemed thinner than normal. They showed a wavy uneveness of density, and all suture lines were wide. The sella turcica was

not clear. There were two tiny metallic-like foreign bodies, which appeared to be within the skull in the posterior parietal area. On the basis of the roentgenographic picture, it was thought that there was increased intracranial pressure and foreign bodies in the skull.

A ventriculogram taken on March 2 showed air in both lateral ventricles and in the third ventricle. They were markedly dilated but centrally placed. No air was seen in the fourth ventricle, and the iter was not definitely outlined.

The impression was that there was a mass in the fourth ventricle.

That same day, through a cross bow incision in the posterior fossa, a tumor which completely filled the fourth ventricle was exposed. On removal, it was found to be adherent to the medulla in the region of the nuclei of Goll and of Burdach.

Case 22.—F. S. V., a woman aged 35, was seen on Oct. 12, 1935, with a history of tinnitus and deafness on the left side for four years. For the past few years there has also been numbness of the left side of the mouth. Later a neuralgic tic of the right fifth nerve developed. For the past year she had

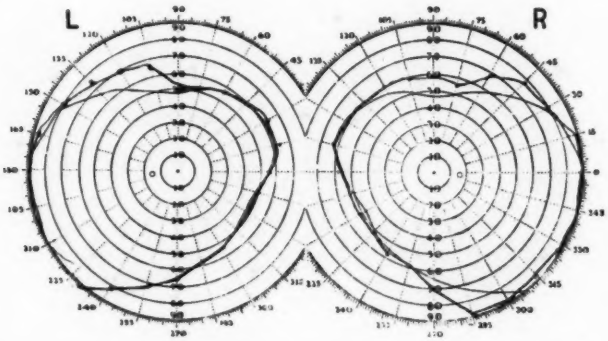

Fig. 23 (case 22).—Visual fields of F. S. V., taken on Oct. 12, 1935, with a 2 mm. white test object at 330 mm.

been having headaches, which for the past four months were fairly constant. Dr. E. J. Browder had made a diagnosis of a tumor of the left cerebellopontile angle.

Ocular examination showed vision of 20/20 in each eye. There was no ocular palsy and no nystagmus. Both disks were swollen about 1.5 D. The veins were distended, but there were no hemorrhages. The visual fields were normal.

The impression was that the visual fields had no localizing value.

On October 16 Dr. Charles Ellsberg, of the Neurological Institute of New York, removed a large meningioma from the left cerebellopontile angle.

Case 23.—J. V., a man aged 43, entered the Brooklyn Hospital on Oct. 28, 1933, with a history of headaches, vomiting and fainting spells for the past three weeks. These began three weeks previously with sudden, severe temporal and occipital headaches and vomiting.

On November 7 a neurologic examination showed the following facts: There was nystagmus only to the right. No personality changes, no loss of memory, no forced grasping and no convulsive seizures or pareses were noted. There

was no astereognosis or loss of two point discrimination and no uncinate seizures. It was advised that nothing be done until more localizing signs developed.

Ocular examination the same day revealed a history of transient attacks of diplopia. The right disk was swollen about 2 D. The margins of the left disk were slightly blurred. The arteries were thin, and the veins were slightly dilated. No hemorrhages or exudates were present. The right visual field shows a concentric contraction, with a defect in the upper temporal quadrant. The left visual field showed a slight concentric contraction.

On the basis of the results of the ocular examination, it was thought that the condition was a tumor deep in the left temporal lobe.

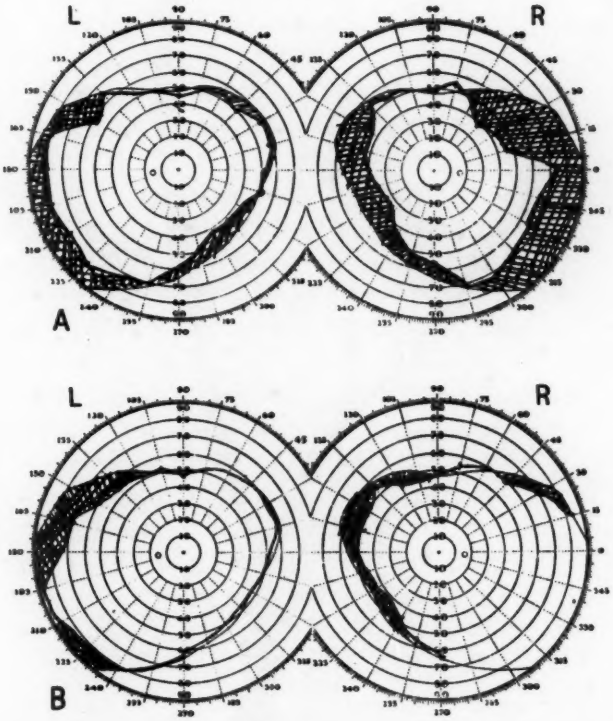

Fig. 24 (case 23).—*A*, visual fields of J. V., taken on Nov. 7, 1933, with a 2 mm. white test object at 250 mm. Vision was 13/13 in each eye. *B*, visual fields of the same patient, taken on November 16, with a 2 mm. white test object at 250 mm. Vision was 13/13 in each eye.

The next day the edema of the right disk had receded, though the margins were still blurred. The left disk showed no change, and there was definite improvement in the visual fields. Because of the improvement, an inflammatory lesion was considered, though a tumor of the brain was not overlooked.

On November 15 the patient became much worse; the nystagmus to the right became more marked and the papilledema increased. Dr. Merwarth therefore suggested the advisability of exploration with the possibility of a tumor of the vermis or the subtentorial region being present.

On November 16 ocular examination showed vision of 13/13 in each eye. The right disk was elevated about 4 or 5 D., and there were several hemorrhages around the disk. The left disk showed only a slight increase in blurring. The visual fields showed no localizing defects.

On November 18 a ventriculogram made by Dr. Browder showed an obstruction at the base of the brain posteriorly, and that day a tumor of the vermis was removed. An extension of the tumor had become adherent to the brain stem and could not be removed.

CONCLUSION

In 1935 Globus [13] analyzed 171 cases of verified tumor of the brain. He concluded that the number of cases in which perimetric study was of diagnostic assistance was surprisingly small, but that when characteristic defects were present they were of immense importance. That the conclusion about the relative small percentage of cases in which visual fields were of diagnostic value was not based on solid premises is shown by an analysis of his series.

Of the 171 cases, no visual fields were taken in 56, or 33 per cent, and visual fields were taken only with the gross finger test in 42, or 24 per cent. But even in the latter group the visual fields showed defects of localizing value in 16 instances. In other words, in 58 per cent of his series of cases either no examination or only a superficial examination of the visual fields was made.

Further analysis shows that in 105 of the cases the tumors were found in the temporal, parietal, occipital and chiasmal regions, situations in which one would expect tumors to give rise to significant localizing signs in the visual fields. In 29 of these cases, or almost 28 per cent, no visual fields were taken, and in 30, or slightly over 28 per cent, visual fields were taken only with the gross finger test. In 29, or 63 per cent, of the remaining 46 cases, the visual fields showed signs of localizing value, and even in 15, or 50 per cent, of the 30 cases in which the visual fields were taken with the gross finger test the fields showed defects of localizing value.

Foster Kennedy, in discussing Globus' paper, expressed the view that the low number of instances in which satisfactory fields could be taken was due to the fact that the studies were undertaken late in the clinical course. This is borne out by the series of cases here presented.

In 5 of the 6 cases of tumor of the frontal lobe, the visual fields, correctly, showed no localizing defects. In the other case (6) the visual fields, incorrectly, showed localizing signs. This patient had good vision, but his mental condition was subnormal and he cooperated poorly.

Tumors of the frontal lobe are also characterized by a paucity of neurologic signs. In 2 of the 6 cases here presented, neurologic examination showed correctly localizing signs; in 2 it showed no findings of localizing value, and in the remaining 2 it showed incorrectly localizing signs, resulting in wrong diagnoses.

Flat roentgenograms were made in 4 of the cases. In 2, the roentgenograms showed defects of localizing value; in 1, no localizing defects and in the other (case 2), defects which resulted in a wrong interpretation.

Encephalograms were made in 4 cases. In 1 case (3) no evidence of tumor was found.

In but 1 of the 8 cases in group 2 (case 11) the visual fields were of no localizing value. The growth in this case was a rare chordoma. It is interesting that the Greens,[14] of San Francisco, described a similar case which practically parelleled the case here reported, even unto the mistakes in diagnoses made by the ophthalmologist, the neurologist and the roentgenologist. In case 12 a wrong interpretation was made. Here again both the vision and the cooperation of the patient were extremely poor. The defects in the visual fields in the other 6 cases were of definite localizing value.

Flat roentgenograms showed no localizing defects in all 4 cases of suprachiasmal lesions but showed definite defects in the other cases.

Correct interpretations were made on the basis of the encephalographic picture in all 6 cases in which encephalography was carried out.

In the third group of 6 cases the visual fields showed, correctly, defects of localizing value. The results of the neurologic examination were of no localizing value in 3 of the cases and gave correct information in the other 3.

Flat roentgenograms showed defects of correct localizing value in only 1 case, while encephalographic examination gave correct information in the 3 cases in which it was carried out.

In the last group of 3 cases, the visual fields, correctly, showed no localizing defects. Neurologic diagnosis was wrong in 1 case (21). Encephalographic examination gave correct evidence in all 3 cases.

In summary, the visual fields in the 14 cases in groups 2 and 3, which would be expected to present localizing defects, showed signs of definite localizing value in 12, or 86 per cent. From a diagnostic standpoint, a correct negative field may be as important to the neurosurgeon as a positive field, as witness cases 3 and 21. Of the 9 cases of tumor in the frontal and the posterior fossa, an incorrect visual field was found in only 1, so that the visual fields in 20 of the 23 cases here presented were correct.

Neurologic examinations, usually made by several observers, gave correct information in 16 cases. In 4 cases the diagnoses were wrong, and in the other 3 neurologic signs were of no localizing value. Flat roentgenograms were taken in 13 cases. In 6, the roentgenographic

14. Green, A. S., and Green, M. I.: Intracranial Chondroma, Bull. Pract. Ophth. 7:85, 1937.

picture was either of no localizing value or gave incorrect information. Encephalograms were made in 15 cases, and in only 1 was the diagnosis missed. However, in 2 cases all other examinations gave incorrect information. The time that elapsed between the onset of symptoms and the diagnoses in those cases in which vision or mentality or both were subnormal varied from three weeks in case 18 to thirteen years in case 10. The longest span of time was in group 2, with an average of six and one half years; the shortest span of time was in group 3, with an average of twenty weeks.

While I do not attempt to say that examinations of the visual fields are of such value that other methods of examination could be dispensed with, I do feel that if done early enough they are of extreme importance. This is especially true in cases of tumor of the pituitary region and of the temporal and the parietal area. If every patient with persistent headache or with other signs suggestive of possible tumor of the brain had an examination of the visual fields, many more instances would be found in which the visual fields are of localizing value. If the visual fields at first are negative and the symptoms persist or are progressive, further studies should be made. It makes no difference if the symptoms are of three weeks' duration or are stretched out over a period of years, visual fields should be taken before blindness or mental deterioration develops. It has been shown how quickly blindness may occur once ocular symptoms develop.

I cannot say that all neurologic examinations should be dispensed with, because in this series they gave correct results in only 70 per cent. Yet I recently looked through the records of a large number of cases of tumor of the brain, and the percentage of cases in which proper visual fields were taken was surprisingly small, about the same percentage as was reported in the series by Globus.

In addition, one should consider the value of positive findings in the visual fields. If the results of the neurologic examination check with findings in the visual fields, it may often make encephalographic or ventriculographic study unnecessary; and for a person with tumor of the brain who is ill, it may mean the margin of safety. However, as shown in case 5, encephalography, when possible, should be carried out.

In closing, I shall say that I feel that examination of the visual fields is an indispensable aid in the diagnosis of tumor of the brain and should be resorted to early and more frequently.

Clinical Notes

RETINITIS PIGMENTOSA WITH "HOLE" IN THE MACULA

Report of a Case

CHARLES A. PERERA, M.D., NEW YORK

Pigmentary degeneration of the retina is a familial and hereditary disease in which the peripheral portion of the retina is most affected. Examples of this condition associated with central retinal changes are uncommon. A search of the literature reveals reports of a number of cases of typical or atypical retinitis pigmentosa combined with disturbances at the macula. Among the writers who have described such cases are Leber,[1] Carruthers,[2] Rieger,[3] Spare[4] and Pavia and Dusseldorp.[5] The ophthalmoscopic picture of a "hole" in the macula in association with retinitis pigmentosa is rare. Leber[6] in his monumental work on diseases of the retina discussed the presence of macular cysts in association with pigmentary degeneration of the retina and referred to cases reported by Hoffman (1885), Meyer (1889), Noll (1908) and Stock (1908). Leber's article included a photomicrograph of a section of the eye of Stock's patient, taken through the macula, showing multiple cysts of the internal nuclear layer of the retina and a large cyst in the fovea.

The patient whose case is reported here was brought to Dr. Charles H. May and has since been under our joint care.

CASE REPORT

Miss A. M. G., an Italian girl of 16, was seen in December 1935, complaining of night blindness and defective peripheral vision for nearly three years. She had consulted several European oculists, who had made the diagnosis of retinitis pigmentosa. Her parents were first cousins. The eyes of her parents

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Presented before the New York Academy of Medicine, Section of Ophthalmology, May 16, 1938.

1. Leber, T.: Ueber anomale Formen der Retinitis pigmentosa, Arch. f. Ophth. **17**:314-341, 1871.

2. Carruthers, J. F.: Case of Retinitis Pigmentosa (Atypical), with Oedema of the Macula, Proc. Roy. Soc. Med. (Ophth. Sect.) **9**:103-105, 1915-1916.

3. Rieger, H.: Ein Beitrag zur Kasuistik der tapeto-retinalen Degeneration, Ztschr. f. Augenh. **57**:429-463, 1925.

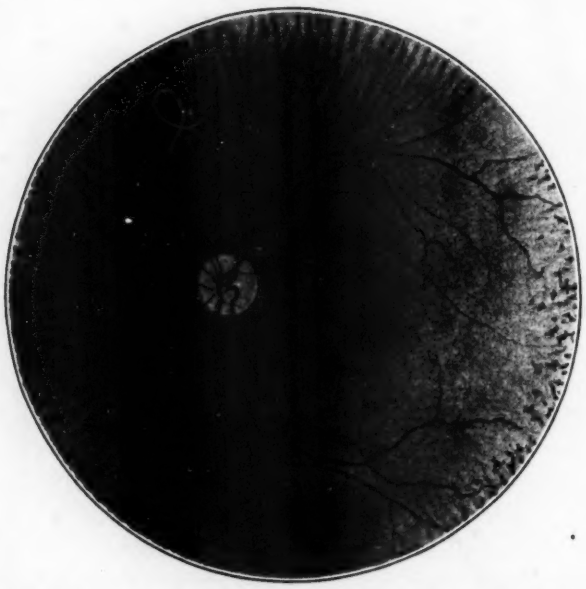
4. Spare, A.: Tapetoretinal Degeneration of the Retina: Case Report, Am. J. Ophth. **11**:545-550, 1928.

5. Pavia, J. L., and Dusseldorp, M.: Degeneracion pigmentaria de la retina con alteraciones de la region macular, Rev. oto-neuro-oftal. **9**:118-125, 1934.

6. Leber, T.: Die Krankheiten der Netzhaut, in Graefe, A., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, 1916, vol. 7, pp. 1680-1682.

and those of her 2 brothers and 3 sisters were normal. The family history was otherwise irrelevant.

Physical examination showed a healthy young girl with no abnormalities except those found in a study of her eyes. Vision was 15/20 in each eye with correcting lenses (right eye, —3.50 sph. —0.75 cyl., axis 15; left eye, —0.75 sph. —0.75 cyl., axis 135). The ocular media were clear except for a few small opacities in the vitreous. Both fundi revealed the typical picture of retinitis pigmentosa, with narrowed vessels and peripheral deposits of pigment. Several small masses of pigment were seen adjacent to the left disk. The optic nerve heads showed limited atrophy, most evident in the left eye. The intraocular pressures and muscle balance were normal. The visual fields with a 3 mm. white test object at 300 mm. were contracted to 20 degrees from the fixation point.



Fundus of the left eye of a patient with retinitis pigmentosa, showing a "hole" in the macula and deposits of pigment adjacent to the nerve head.

During the following year and a half, the patient was seen at intervals of from four to six months, with no changes in the ocular picture. She was attending school and planned to enter college.

In June 1937 she reported to our offices, complaining of blurred and distorted vision of the left eye for several weeks. Examination showed the vision of this eye to have been reduced to 15/60. Examination of the left fundus revealed slight edema of the retina around the macula and a round faint reddish area in the fovea, suggestive of a beginning "hole." There was a small central scotoma in the field of the left eye. The right eye and the peripheral fields of both eyes were unchanged. Careful questioning elicited no history of trauma. Search for foci of infection was fruitless. The results of all laboratory tests, including a Wassermann test of the blood, were negative.

Within three months the vision of the left eye decreased to 15/400. The lesion in the left fovea took on the characteristic appearance of a hole, which has increased in size to a slight extent during the six months preceding the

writing of this report. The right eye has remained unchanged. The visual fields now show a large absolute central scotoma in the left eye but no further contracture. The accompanying photograph of a painting of the fundus by Mr. Bethke depicts the lesions of the left fundus in December 1937.

COMMENT

The frequency of macular involvement in association with diseases of the retina is explained by the delicate and highly organized structure of this region and by the sluggish circulation due to absence of blood vessels. These factors make the macula particularly vulnerable to toxic and circulatory disturbances.

The so-called hole of the macula represents in most instances the ophthalmoscopic appearance of conglomerate or confluent cysts and has been reported to occur following contusion of the eyeball, penetrating injury of the globe, detachment of the retina, intraocular inflammation and vascular disease. The typical macular "hole" develops from cystic degeneration and is rarely the result of a retinal tear.

Most authors believe that the cystic changes leading to the formation of a "hole" have their origin in retinal edema and in the disappearance of retinal elements from insufficient nutrition. The pathologic picture of macular "holes" has been reviewed by Coats,[7] Samuels [8] and Reese.[9] The last-named writer stated: "There may be cystic formations in the macula, usually in the early stages of the disease (retinitis pigmentosa). These may be transitory and are usually attributed to edema."

Schieck [10] divided diseases of the macula into those due to extramacular processes (injuries, inflammations, glaucoma, myopia, thrombosis of the central vein of the retina, etc.) and those which occur without extramacular disease. He further subdivided the latter group into hereditary and senile or atherosclerotic diseases, including among the hereditary diseases the spontaneous, bilateral condition which Behr had described under the title of heredodegeneration of the macula (congenital, juvenile, adult, presenile and senile).

Pigmentary degeneration of the retina and macular dystrophies are classified together by many writers under the progressive dystrophies or abiotrophies. The latter term was coined by Gowers and refers to premature senility of the involved tissue due to defective vitality. Treacher Collins [11] wrote at length on the abiotrophic origin of retinitis pigmentosa, although the discussers of his paper (Mayou, Greeves,

7. Coats, G.: The Pathology of Macular Holes, Roy. London Ophth. Hosp. Rep. **17**:69-96, 1907.

8. Samuels, B.: Cystic Degeneration of the Retina, Arch. Ophth. **4**:476-486 (Oct.) 1930.

9. Reese, A. B.: Defective Central Vision Following Successful Operations for Detachment of the Retina, Am. J. Ophth. **20**:591-598, 1937.

10. Schieck, F., in Henke, F., and Lubarsch, O.: Handbuch der speziellen Pathologie, Anatomie und Histologie, Berlin, Julius Springer, 1928, pt. 1, pp. 658-667.

11. Collins, E. T.: Abiotrophy of the Retinal Neuro-Epithelium or "Retinitis Pigmentosa," Tr. Ophth. Soc. U. Kingdom **39**:165-195, 1919.

A. Critchett, Morax and Parsons) expressed the opinion that vascular disease of the retina and choroid played a major role. Mann [12] described retinitis pigmentosa as a latent fault leading to the appearance of a postnatal degenerative process and related genetically to lesions of various regions of the fundus (including macular degeneration). She looked on both retinitis pigmentosa and macular degeneration as regional failures, with abnormal ectodermal and mesodermal elements.

The formation of a macular "hole" has been watched by many ophthalmologists, but only a few have reported their observation of the developing lesion. Crawford [13] described an instance of this. I have recently studied the fundi of 2 elderly persons with senile degeneration of the macula and have watched the formation and development of a typical "hole" in one eye of each patient.

In the absence of more definite knowledge of the causation of retinitis pigmentosa and juvenile macular degeneration, it seems logical to consider the two processes described in this paper as due to the effects of vascular and nutritional disturbances on a congenitally abnormal and defective retina. If this is true, one must accept the possibility of future central degenerative changes in the retina of the eye with the uninvolved macula.

SUMMARY

Examples of retinitis pigmentosa associated with macular changes are uncommon. Instances in which a "hole" occurs in the macula are exceedingly rare. This combination of conditions is reported in a 16 year old Italian girl whose parents were first cousins. The macular lesion developed in one eye while the patient was under observation. I believe that the peripheral and central degenerative lesions in the retina of this patient were the results of local vascular disease and retinal deterioration. The relevant literature is reviewed.

12. Mann, I.: Developmental Abnormalities of the Eye, Cambridge, England, Cambridge University Press, 1937.

13. Crawford, J. W.: Hole in the Macula: Report of a Case in a Patient Under Observation, Arch. Ophth. **10**:793-799 (Dec.) 1933.

HYPERSENSITIVITY TO LAROCAINE

FREDERICK H. THEODORE, M.D., NEW YORK

Larocaine hydrochloride, *p*-aminobenzoyl-2, 2-dimethyl-3-diethylaminopropanol hydrochloride, has gained considerable popularity as a local anesthetic in the United States and abroad in the past five years. It has been used successfully for many surgical procedures, particularly for those in the fields of otolaryngology and ophthalmology.[1] It is used satisfactorily in a 2 to 5 per cent solution for instillation into the eye.[2]

1. Rich, B.: Larocaine: A Summary, M. Rec. **144**:419-423 (Nov. 4) 1936.

2. Mayer, L.: Larocaine: A New Anesthetic, Arch. Ophth. **14**:408-411 (Sept.) 1935.

For subcutaneous injection, solutions varying in strength from 1 to 10 per cent have been used. Up to the present no ill effects from this drug have been reported. Because of this, it was thought worth while to report the following case of hypersensitivity.

REPORT OF CASE

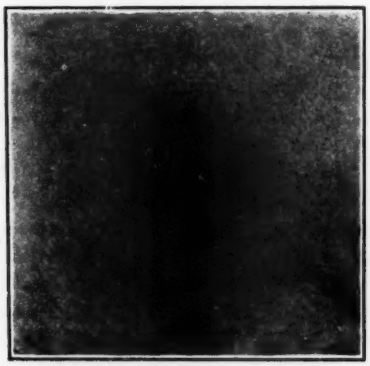
M. T., a woman aged 70, was first examined in May 1937 because of opacities of the vitreous which had been annoying her. She had a high degree of myopia. Mild chronic conjunctivitis was noted as an incidental finding. Her past history was unimportant, except that in November 1934, during the course of a tineal infection of the skin, at the time unrecognized, she had undergone complete allergic tests and had been found sensitive only to chocolate. Six years previously a small dose of sodium barbital (soluble barbital U. S. P. XI) had given her a rash.

She returned on December 2, because of acute conjunctivitis in each eye. Smears of conjunctival material revealed no organisms. Treatment consisted of the topical application of a 1 per cent solution of silver nitrate preceded by the instillation of a 2 per cent solution of larocaine hydrochloride. The patient was instructed to use a 25 per cent solution of mild protein silver at home. She returned one week later, considerably improved but not entirely well. A 1 per cent solution of silver nitrate, preceded by anesthetization with a 2 per cent solution of larocaine hydrochloride, was again applied. When the patient was next seen it was apparent that she was much worse. A number of fine punctate superficial corneal infiltrations had appeared in each eye, which stained with fluorescein. Again a solution of silver nitrate was used after anesthetization with larocaine hydrochloride. In the course of a week, during which almost daily treatments first with silver nitrate and then with the light application of a copper stick were given, each time after anesthetization with larocaine hydrochloride, the condition became increasingly worse. Finally, there was considerable massive edema of the eyelids and the upper half of the face, which, combined with swelling of the parotid glands due to stenosis of the parotid ducts, gave a leonine appearance to the patient. Both the bulbar and the palpebral conjunctiva were now diffusely injected, and there was much mucopurulent discharge, which was free from organisms. In the left eye, at the limbus, there was a complete ring of marginal infiltration of the cornea due to the coalescing of discrete catarrhal ulcers. In the right eye the marginal ulcers were less extensive. The corneal epithelium of each eye showed myriads of fine punctate infiltrations.

It was apparent that the treatment was aggravating the condition, and at this stage, fortunately, medication was stopped, and the patient was instructed to use a mild eyewash. In a few days she was much better, and within five days all the corneal lesions had disappeared. Thereafter, applications of a mild solution of silver nitrate, from 0.16 to 0.33 per cent, without the previous use of larocaine hydrochloride, were employed with benefit. On Feb. 10, 1938, in the course of examining the right lacrimal passage, 1 drop of a 2 per cent solution of larocaine hydrochloride was instilled into the right eye only. Within a few hours the eye began to bother the patient, and twenty-four hours later both the bulbar and the palpebral conjunctiva were found to be diffusely inflamed, and two catarrhal corneal ulcers the size of a pinhead were present. Again, with the use of mild eyewashes the condition cleared rapidly.

It now appeared clinically certain that hypersensitivity to larocaine had been responsible for the entire disturbance from the beginning. Patch tests were made

with 10 per cent larocaine hydrochloride in an oxycholesterol petrolatum ointment base. There was no itching till twenty-four hours had elapsed. When the patch was removed after forty-eight hours, the skin was erythematous and indurated, and numerous vesicles were observed. Control tests with the ointment base and several other anesthetic ophthalmic ointments were negative. The sensitized skin became more inflamed, and the epidermis sloughed. One month later it had not entirely healed. All the previous solutions of larocaine hydrochloride and the ointments containing the drug had been made from the commercial tablets. In order to rule out any other factor, the patch tests were again done with pure crystalline larocaine hydrochloride.[3] This time ointments containing 0.5 and 0.1 per cent larocaine hydrochloride were used. With 0.5 per cent ointment there was a markedly positive reaction in forty-eight hours, with much vesiculation and, later, epidermal sloughing. This was even more marked than with the ointment which contained 10 per cent larocaine hydrochloride. Even the 0.1 per cent ointment produced a little erythema and then a few vesicles. These patch tests confirmed



Positive reaction after seventy-two hours to a patch test on the inner aspect of the arm with the 0.5 per cent larocaine ointment.

the clinical impression that an increasing sensitivity to the drug had been occurring.

COMMENT

Hypersensitivity to larocaine hydrochloride must be unusual, and its rare occurrence should not prejudice its position as an excellent anesthetic. Similar anesthetics likewise at times cause unpleasant reactions.[4] Had it been realized, however, that this drug is capable of producing untoward effects, considerable suffering might have been prevented. Moreover, the occurrence of constantly increasing sensitivity during the period of its administration may be of importance and should be noted. An appreciation of these facts is even more important in those cases in which larocaine hydrochloride may be used as an infiltration anesthetic, which, conceivably, might give rise to more serious consequences.

3. This was supplied by Hoffmann-La Roche, Inc.

4. Pfeiffer, R.: Hypersensitivity to Pontocaine, Arch. Ophth. **18**:62-64 (July) 1937.

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

BINOCULAR VISION AND ORTHOPTIC PROCEDURE

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NEW YORK

The clinical application of orthoptic procedure in the treatment of imbalance of the ocular muscles has led to the publication of various reports which, because of differing criteria of success, incomplete records and inadequate control groups, show inconsistent results and justify skepticism as to the legitimate place of orthoptic training in ophthalmology. Indiscriminate use of ocular exercise by unqualified persons has contributed to the popular misconception of orthoptic procedure as a substitute for glasses or surgical treatment rather than a supplementary technic useful to the ophthalmologist for the diagnosis and treatment of defects of binocular vision. The physiologic basis for binocular vision and the clinical diagnosis of its anomalies will be briefly reviewed as a preface to a more detailed discussion of actual orthoptic procedure, which may contribute to more careful clinical work and at the same time indicate the need for controlled laboratory research in this field.

THE BASIS OF BINOCULAR VISION

Phylogenetically, binocular vision is correlated with the forward placement of the eyes, evident first in carnivora, giving overlapping fields of vision. Semidecussation rather than complete crossing of the optic fibers at the chiasm, giving bilateral cerebral representation of the eyes in higher forms, occurs in such a manner that the right portion of each retina is represented in the right cerebral hemisphere, while the left portion of each retina is represented in the left hemisphere.[1] Ontogenetically, in the prenatal development of the human eyes, the angle between the optic axes is reduced from 180 degrees to about 60 degrees.[2] The neurologic foundation for bimacular fixation is present from birth, and though meager experimental evidence indicates a gradual develop-

From the Orthoptic Clinic of the Manhattan Eye, Ear and Throat Hospital.

1. Adler, F. H.: Clinical Physiology of the Eye, New York, The Macmillan Company, 1933, p. 230.

2. Mann, I.: Development of the Human Eye, Cambridge, University Press, 1928, pp. 43 and 264.

ment of coordinate movements during the first year, one cannot assume an arbitrary age at which binocular vision in all its aspects has reached maturity.

Assumptions concerning the exact projection of the visual fibers in the occipital cortex have been based on evidence derived from defects in the visual field and localized central lesions resulting from gunshot wounds and other injuries. Rearrangement of the fibers in the lower part of the optic tracts is believed to take place in such a way that fibers from corresponding parts of the two retinas lie near each other,[3] but since the time of Sherrington [4] it has been assumed that fusion occurs in the final projection areas of the cortex. The typical sparing of the macula in severe defects of the visual field has given support to the widely accepted theory that the macular fibers have bilateral cortical representation, though Fox and German [5] demonstrated widespread distribution of the macular fibers, which may make it unnecessary to postulate bilateral macular projection to account for such phenomena.

The earliest theories of fusion assumed actual connection of nerve fibers in or near the chiasm or alternation of the two eyes, so that one eye at a time actually responds to visual stimulation.[6] Müller's postulation of "corresponding points," [7] which were assumed to have anatomically identical projection fibers, was widely accepted, though it is evident from simple demonstrations of physiologic diplopia that numerous double images must be suppressed at all times. Anatomic evidence of point to point correspondence in the central projection areas has never been shown. Moreover, Peckham [8] proved that single vision is maintained when images are far from "corresponding" points. Henschen [9] demonstrated that early loss of both eyes results in atrophy of both the superficial and the deep layer of cells in the area striata, leaving the middle, functionally independent layer intact, whereas early loss of one eye results in atrophy of the deep layer of cells on the opposite side of the brain and the superficial layer of cells on the same side of

3. Adler,[1] p. 155.

4. Sherrington, C. S.: Integrative Action of the Nervous System, New Haven, Conn., Yale University Press, 1911, p. 381.

5. Fox, J. C., Jr., and German, W. J.: Macular Vision Following Cerebral Resection, Arch. Neurol. & Psychiat. **35**:808 (April) 1936.

6. Helmholtz, H. L. F.: Helmholtz's Treatise on Physiological Optics, English translation, edited by J. P. C. Southall, Rochester, N. Y., The Optical Society of America, 1925, vol. 3, p. 482.

7. Müller, J.: Zur vergleichenden Physiologie des Gesichtsinnes des Menschen und der Thiere, Leipzig, C. Cnobloch, 1826.

8. Peckham, R. H.: Foveal Projection During Ductions, Arch. Ophth. **12**: 562 (Oct.) 1934.

9. Henschen, S. E.: Zur Anatomie der Sehbahn und des Sehzentrums, Arch. f. Ophth. **117**:403, 1926.

the brain. Thus the middle layer of cells, bordered by cells connected with functionally related parts of each eye, is probably essential for fusion. These cells are fully developed at birth.

Verhoeff [10] proposed a theory of binocular vision in which he accepted the assumption of corresponding points, which are presumably represented anatomically in the occipital lobes in close proximity, yet separately. He presented convincing evidence from stereoscopic charts that one eye is dominant for some parts of the visual field while the other is dominant for other parts, so that single impressions from corresponding retinal images are really made up of parts of one image and parts of the other or, in some cases, all of one image or all of the other. In stereopsis, in which there is apparent union of disparate images (those falling on noncorresponding retinal areas), Verhoeff demonstrated that one of the disparate images is completely replaced by its substitute without disturbing the effect of depth.

In addition to the empiric factors in the judgment of distance which operate for monocular vision alone, such as apparent size, motion, interposition, shadows, perspective and parallax by movements of the head, and possibly accommodation, in normal binocular vision the factor of binocular parallax, based on disparate images and enhancing the appearance of depth, acts for finite distances.[11] True binocular vision requires the presence of bifoveal fixation, a binocular field mediated by uncrossed retinal fibers, similar retinal images and approximately equal acuity in the two eyes.[12] Stereopsis is precluded by a difference in visual acuity when the ratio amounts to more than 6/6 to 6/9 according to Adler,[13] though Verhoeff [14] suggested a kinetic test for stereoscopic vision which is successful when the acuity is as low as 6/200 in one eye or in both eyes.

The maintenance of binocular vision in various fields of fixation and at various distances is dependent on the normal functioning and coordination of the intraocular and extraocular muscles. Cortical centers correlated with voluntary ocular movement have been demonstrated by electrical stimulation of the exposed cortex of the frontal lobes; centers for involuntary ocular movement, induced by visual stimuli, are situated in the occipital lobe.[15] The extrinsic ocular muscles have been shown by experiment to exhibit the phenomenon of reciprocal innervation, the

10. Verhoeff, F. H.: A New Theory of Binocular Vision, Arch. Ophth. **13**: 151 (Feb.) 1935.

11. Helmholtz,[6] p. 371.

12. Adler,[1] p. 229.

13. Adler,[1] p. 241.

14. Verhoeff, F. H.: Kinetic Test for Stereoscopic Vision, Arch. Ophth. **15**: 835 (May) 1936.

15. Adler,[1] p. 294.

contraction of one muscle being accompanied by simultaneous relaxation of its antagonist. The third, fourth and sixth cranial nerves, controlling the ocular muscles, are subject to the all or none law, the size of the response being dependent on the number of stimulated fibers in each motor nerve.[16] The neural center for convergence, a disjunctive ocular movement, is assumed to be the median group of cells (Perlia's nucleus) in the nucleus of the third nerve;[17] no anatomic center for divergence has been demonstrated. In emmetropic eyes, which are orthophoric, the amount of convergence in meter angles[18] is theoretically equivalent to the amount of accommodation in diopters. There is evidence that the two functions, innervated by the third nerve, have become anatomically interrelated by the process of neurobiotaxis,[19] so that accommodation causes simultaneous convergence. That the two functions can be dissociated to some extent is evident from the fact that refractive errors which increase or decrease the amount of accommodation necessary for clear vision may be present without disturbing the coordination of convergence. Nevertheless, complete dissociation of the two functions is not physiologically possible, and the application of orthoptic training is limited by this fact.

Worth's arbitrary classification of grades of fusion, based on artificial criteria, is in general use in discussions of binocular vision.[20]

1. First grade fusion: simultaneous bimacular perception. (This grade of fusion has been subdivided by some clinicians into two types: simultaneous perception without superposition of images and simultaneous perception with superposition of images.[21] It is doubtful if bimacular perception without superposition, i. e., in which the observed images are widely separated subjectively, can be called fusion in the usual sense.)

2. Second grade fusion: simultaneous bimacular perception with effort to maintain fusion by ocular movements when fixation is changed ("amplitude" of fusion).

3. Third grade fusion: stereopsis, or sense of depth mediated by binocular cues.

ANOMALIES OF BINOCULAR VISION

Orthophoria, or normal muscle balance, like emmetropia, is the exception, since careful tests reveal heterophoria in over 80 per cent of cases,

16. Adler,[1] p. 301.
17. Adler,[1] p. 298.
18. Adler,[1] p. 274.
19. Stutterheim, N. A.: Indications for the Kinetic Treatment of the Eyes, London, G. Pulman & Sons, 1931, p. 10.
20. Worth, C.: Squint, ed. 5, London, Baillière, Tindall & Cox, 1921, p. 12.
21. Lyle, K., and Jackson, S.: Practical Orthoptics in the Treatment of Squint, London, H. K. Lewis & Co., Ltd., 1937, chap. 7.

according to Bielschowsky.[22] Clark [23] found deviations from normal for the near point in 90 per cent of 191 college students tested. Mild degrees of muscle imbalance which cause no symptoms may be considered within normal limits. According to Berens, Losey and Hardy,[24] deviations for distance should not exceed 3 diopters of esophoria, 2 diopters of exophoria or 1 diopter of hyperphoria; deviations for the near point should not exceed 1 diopter of esophoria, 6 diopters of exophoria or 1 diopter of hyperphoria. Heterophoria, or latent deviation, demonstrable only by tests which dissociate the eyes by preventing fusion, probably precedes heterotropia, in which there is a manifest deviation, frequently called "strabismus" or "squint." The constant innervation necessary to maintain binocular vision in the presence of heterophoria may give rise to marked subjective symptoms, whereas no effort is made to maintain binocular vision in the presence of heterotropia, and symptoms consist of cosmetic defect or low visual acuity. The causes of nonparalytic strabismus have offered a fertile field for speculation, and present day treatment assumes no single factor, such as refractive error or fusion deficiency, as responsible for the defect.

No essential neurologic difference between monolateral and alternating strabismus has been demonstrated. That there is probably a close relation between the two types is evident from the fact that many persons who have alternating fixation at first later have a strong preference for monolateral fixation and persons who have monolateral fixation can be taught to alternate. Clinicians are agreed that patients with spontaneous alternating fixation, with equal acuity in the two eyes and, frequently, insignificant refractive errors, present greater difficulties in correction by optical, surgical or orthoptic methods than those who have a strong preference for monolateral fixation. It has been suggested that alternating fixation is accompanied by more firmly established functional changes [25] than those which accompany monolateral strabismus, but there may be central neurologic factors as yet unobserved which contribute to alternating fixation and interfere with successful treatment.

Significant functional changes which occur in cases of strabismus are: suppression, defects in the visual field, amblyopia ex anopsia and anomalous projection. All of these factors operate to facilitate single vision and prevent diplopia, which interferes with accurate orientation. Sup-

22. Bielschowsky, A.: Functional Disturbances of the Eyes, Arch. Ophth. **15**:589 (April) 1936.

23. Clark, B.: Heterophoria in College Students, Am. J. Optometry **12**:9, 1935.

24. Berens, C.; Losey, R. L., and Hardy, L. H.: Routine Examinations of the Ocular Muscles and Nonoperative Treatment, Am. J. Ophth. **10**:910, 1927.

25. Lyle and Jackson,[21] p. 114.

pression characteristic of strabismus is generally assumed to be a central process, though there is some evidence that peripheral mechanisms may contribute to suppression. Fry [26] demonstrated that voluntary suppression is accomplished by changes in accommodation, and Javal [27] pointed out that in cases of strabismus the deviating eye loses its ability to accommodate accurately for a given point of fixation, and this inaccuracy may simulate a considerable degree of amblyopia. Suppression scotomas have been mapped by Travers in cases of strabismus; [28] and Braun [29] showed that an absolute scotoma to the temporal side of the fixation spot can be found in cases of monolateral fixation, whereas the fields are normal in cases of alternating strabismus. Prolonged and constant suppression is assumed to cause amblyopia ex anopsia. Most important of the functional changes associated with strabismus of long standing is anomalous projection ("abnormal retinal correspondence," "secondary retinal correspondence," "incongruous retinas" or "false projection"), in which the foveal perception of the fixing eye is associated with peripheral perception of the deviating eye in an inferior type of binocular vision. When both eyes are in use, the projection of the deviating eye is interpreted as if the peripheral image were "fused" with the foveal image of the fixing eye and projected to the same point in space. Acuity of the peripheral region used under such conditions by the deviating eye does not increase above that to be expected in a peripheral retinal area. Except in rare cases of eccentric fixation, sometimes called "false macula," fixation returns to the foveal region in the deviating eye while the fixing eye is covered, even in cases of well established anomalous projection, so that the screen test remains a valid method of measuring the absolute deviation. It should be emphasized that anomalous projection is a binocular phenomenon, alteration in projection occurring for the entire field, as pointed out by Maddox.[30] The presence of anomalous projection invalidates measurements made with subjective tests (Maddox rod) in which there is no control of foveal fixation.

In anomalous projection, bifoveal stimulation, possible under artificial conditions, results in paradoxic diplopia: heteronymous diplopia in

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26. Fry, G. A.: The Relation of Accommodation to the Suppression of Vision in One Eye, Am. J. Ophth. **19**:135, 1936.

27. Javal, L. E.: Du strabisme dans ses applications á la physiologie de la vision, Paris, Victor Masson et Fils, 1868, p. 53.

28. Travers, T. B.: Concomitant Strabismus, London, G. Pulman & Sons, 1936, p. 46.

29. Braun, G.: Gesichtsfelduntersuchungen bei Schielenden, Klin. Monatsbl. f. Augenh. **42**:600, 1934.

30. Maddox, E.: The Ocular Muscles, ed. 2, Philadelphia, Keystone Publishing Company, 1907, p. 125.

convergent deviations, and homonymous diplopia in divergent deviations. Von Graefe [31] reported postoperative paradoxic diplopia in 1854; it can easily be demonstrated preoperatively by controlled bifoveal fixation. Tschermak [32] and others have used the projected foveal afterimages as a test for anomalous projection.

It is probable that innate primary retinal correspondence is seldom replaced completely by secondary retinal correspondence since normal projection with bifoveal fixation eventually develops in many cases of postoperative paradoxic diplopia. Moreover, anomalous projection is frequently unstable, considerable variation in the angle of the anomaly (the position of peripheral fixation) being observed on repeated tests, though von Kries [33] stated that "in many instances the anomalous visual relation is a comparatively fixed one, strongly prevailing over the original, if the latter is still present at all."

Verhoeff [34] stated that he did not believe that there could be any retinal correspondence in anomalous projection and criticized the usual methods for demonstrating anomalous projection because of the highly artificial conditions set up for their use.

ORTHOPTIC PROCEDURE

Scattered references to orthoptic procedure can be found in the ophthalmologic literature for the last forty years without complete descriptions of the exercises used or the exact methods employed. Recently, more practical information concerning the technic of orthoptic training has been published, since the directions furnished with instruments are seldom adequate and courses in orthoptics, such as those given in several British clinics, are not generally available.

Worth's book on squint [35] in 1903 revived interest in the subject of orthoptics, which Javal [36] had treated in 1896. Wells' [37] emphasis on the use of the stereoscope in ophthalmology and the improvements in orthoptic instruments gave further impetus to the testing and training of binocular vision. Textbooks of ophthalmology mention the subject only

31. von Graefe, A.: Ueber das Doppelsehen nach Schieloperationen und Incongruenz der Netzhäute, Arch. f. Ophth. **1**:82, 1854.

32. Tschermak, cited by Helmholtz,[6] p. 588.

33. von Kries, cited by Helmholtz,[6] p. 588.

34. Verhoeff, F. H.: Anomalous Projection and Other Visual Phenomena Associated with Strabismus, Arch. Ophth. **19**:663 (May) 1938.

35. Worth, C.: Squint, Philadelphia, P. Blakiston's Son & Co., 1903.

36. Javal, E.: Manuel théorique et pratique du strabisme, Paris, G. Masson, 1896.

37. Wells, D. W.: The Stereoscope in Ophthalmology, ed. 2, Boston, Globe Optical Company, 1918.

briefly, but in 1932 Cantonnet and Filliozat [38] published their book on strabismus and its reeducation, an English translation of which has appeared.[39] British contributors have been foremost in the publication of articles and textbooks on orthoptic practice. The works of Dobson,[40] Pugh[41] and Lyle and Jackson [21] may be referred to for descriptions of instruments and their use in orthoptics; Berens' "The Eye and Its Diseases" [42] includes an article on orthoptics by Pugh; Travers [28] attempted to evaluate the place of orthoptics in the treatment of concomitant strabismus. Articles in periodicals usually are confined to clinical studies without actual detail of orthoptic practice, and frequently incomplete records make evaluation of the studies difficult.

Orthoptic tests can be carried out satisfactorily only if the proper surroundings contribute to quietness, isolation and unhurried examinations. Individual supervision of testing and training is essential even when mechanical instruments are employed. Half-hour periods several times weekly are usually recommended. Cantonnet [43] suggested one office visit weekly with a full hour of home work daily. Daily clinical work is usually impossible because of limited facilities, but fewer than three visits weekly, when no home work can be given, is hardly adequate to constitute "training."

No systematic studies of strabismic children for comparison with normal children in motor skills, personality traits, intelligence, scholastic achievement or social adjustment have been published. Lippmann [44] found no evidence of more frequent stammering or lefthandedness in strabismic children; von Csapody-Mócsy [45] showed that children having convergent strabismus had symptoms of rickets more frequently than a general group of the same population. Dor [46] suggested a possible relation between vitamin A deficiency and the development of strabismus

38. Cantonnet, A., and Filliozat, J.: Le strabisme, sa reeducation, Paris, Norbert Maloine, 1932.

39. Cantonnet, A., and Filliozat, J.: Strabismus: Its Reeducation, translated by M. Coque, Chicago, M. Wiseman & Company, 1934.

40. Dobson, M.: Binocular Vision and the Modern Treatment of Squint, New York, Oxford University Press, 1933.

41. Pugh, M. A.: Squint Training, New York, Oxford University Press, 1936.

42. Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936, p. 908.

43. Cantonnet and Filliozat,[39] p. 302.

44. Lippmann, O.: Die Entartungszeichen und die Ueberwertigkeit einer Körperhälfte in ihrer Bedeutung für das Schielen, Klin. Monatsbl. f. Augenh. **92**:370, 1934.

45. von Csapody-Mócsy, M.: Ein Beitrag zur Pathologie des Begleitschielens, Klin. Monatsbl. f. Augenh. **92**:385, 1934.

46. Dor, L.: Strabisme et avitaminose, Arch. d'opht. **50**:667, 1933.

when a hereditary predisposition exists. Dull children are frequently poor subjects for orthoptic training, as their attention span is limited and their interest difficult to maintain. Cantonnet,[47] believing that the best results are to be expected from older children, accepted no patients under 7 years of age for orthoptic training. Accurate results in the testing of visual acuity by means of illiterate charts can usually be obtained when a child is between 3 and 4 years of age, and tests of binocular vision can be tried before a child is 2 years of age, though satisfactory tests require sufficient vocabulary to name simple objects. The age at which orthoptic training can be attempted depends on intellectual maturity and cooperation rather than on chronologic age.

Minimum orthoptic equipment should include a major amblyoscope with proper charts for measuring deviations and grading binocular vision as well as for duction training; supplementary fusion tests, and materials for visual training. The advantage of a major amblyoscope over the simple stereoscope lies in greater flexibility, easier control of macular fixation and greater convenience for rapid and accurate testing of binocular vision. In clinical work, additional equipment may be indicated, but the more complicated instruments employing moving targets have limited usefulness. Unilluminated stereoscopes and the simple amblyoscope are not readily employed to determine the type of projection used by the patient, since the examiner should have an unobstructed view of the subject's eyes during tests. The major amblyoscopes, like the simple stereoscope, contain convex spheres to give optical infinity at the distance chosen for the charts employed; prismatic effects are obtained by changing the actual position of the reflected images. Accommodation may be stimulated by the addition of concave spheres for near point tests or, in some instruments, by moving the charts closer to the eyes. Accurately prepared charts are necessary for test purposes, but appropriate slides for duction training can be cheaply constructed by mounting tissue pictures in standard slide glass covers or tracing crayon pictures on etched glass. The angular size of the images can be determined by the use of the proper formulas; standard charts may be graded in angular size.

Complete records for orthoptic training are essential and should include general examination and preliminary treatment as well as accurate information concerning clinical visits and home work. A convenient record form for clinical use which can be printed on one side of standard 8½ by 11 inch (21 by 28 cm.) sheets, or on 5 by 8 inch (12 by 20 cm.) record cards if both sides are used, is shown in the accompanying table.

47. Cantonnet and Filliozat,[89] p. 228.

Orthoptic Record

Number.....	Clinic.....	Development.....	Handedness.....
Name.....	Age.....	Diseases.....	I. Q. .....
Address.....	Birth.....	Operations.....	Cooperation.....

Strabismus, type.....	Deviation:	Previous treatment:
Heredity.....	Convergent.....	Glasses.....
Age of onset.....	Divergent.....	Occlusion.....
Mode of onset.....	Vertical.....	Atropine.....
Cause.....	Occasional.....	Orthoptics.....
Symptoms.....	Periodic.....	Operation.....
Fixing eye.....	Constant.....	

Examination:	Refraction and Prescription			Binocular Vision	Dates
Fundus.....	Date	Cyclo-plegic	Static	Rx.	Projection.....		
Media.....					Suppression.....		
Fixation.....	1				Fusion		
Motility.....					Distance.....		
Fields.....	2				Near.....		
Amblyopia.....					Amplitude		
	3				Distance.....		
Head position.....					Near.....		
Angle gamma.....	4				Worth dots		
P. D.*.....					Distance.....		
PcB.†.....					Near.....		
					Stereopsis.....		

Operations:

Recommendations:

Date	Time	Acuity		Deviation				Amplitude		Notes	Home Work	Return
				Screen		Synoptophore						
		Cc.‡	Sc.§	Far	Near	Obj.	Subj.	Ad.	Ab.			

* Interpupillary distance.
† Near point of convergence.
‡ With correction.
§ Without correction.

In preliminary treatment of heterophoria and heterotropia, the significance of accurate correction of refractive errors cannot be overemphasized. Superficial reports of the "success" of orthoptic treatment frequently fail to indicate the importance of proper optical correction in aiding the coordinate action of the eyes, and it is probable that in many cases the correct prescription is of far greater effective value than orthoptic exercise. The value of any method of visual reeducation or coordinate exercise applied before optical correction of refractive errors has been used constantly for a period of several months is certainly open to question because spontaneous improvement in both acuity and coordination may occur without recourse to special training. No careful study of the length of the period during which correcting lenses should be worn previous to orthoptic treatment has been made. Guibor [48] and Bressler [49] suggested the wearing of the proper optical correction for

48. Guibor, G. P.: The Possibilities of Orthoptic Training, Am. J. Ophth. **17**:835, 1934.

49. Bressler, J. L.: Orthoptic Treatment of Strabismus, Illinois M. J. **68**: 273, 1935.

six months before orthoptic training. Fuchs (Duane)[50] pointed out that the wearing of correcting lenses does not give an immediate effect: "It usually takes some months for the full effect of the glasses to be secured." Maddox[51] stated: "The correction of the refraction does not always cure an accommodative squint at once, but lessens it by degrees." Parinaud[52] recommended optical treatment for at least six months previous to other treatment. Feldman[53] accepted patients for orthoptic training after glasses have been worn six weeks with no improvement. Cords[54] in a statistical summary indicated that over 80 per cent of persons with convergent concomitant strabismus have hypermetropia, and since this defect carries with it the necessity for excessive accommodation, lenses which relieve this should certainly be worn before other treatment. Guibor[55] found that 23.6 per cent of 185 persons with convergent strabismus responded to correction of the refractive error alone. Of a group of 377 persons with convergent strabismus studied at the Manhattan Eye, Ear and Throat Hospital,[56] 26.25 per cent responded to correction of refractive errors without other treatment.

Most authorities recommend for children the use of a 1 per cent solution of atropine two or three times daily for at least three days before retinoscopic examination. For persons with strabismus, Thorington[57] recommended the daily use of atropine for two or three weeks; Howe[58] stated that in the nonoperative treatment of strabismus accommodation should be relaxed for months. Parinaud[59] recommended the use of atropine for at least a week previous to tests; Javal[60] used atropine twice daily for one week, with atropine and cocaine used three times, three hours before examination. Cords[61] recommended the use

50. Fuchs, E.: Textbook of Ophthalmology, ed. 8, translated by A. Duane, Philadelphia, J. B. Lippincott Company, 1924, p. 335.

51. Maddox,[30] p. 119.

52. Parinaud, H.: Traitement du strabisme, Bull. et mém. Soc. franç. d'opht., 1893, p. 50.

53. Feldman, J. B.: Orthoptic Treatment of Concomitant Squint, Arch. Ophth. **13**:419 (March) 1935.

54. Cords, R.: Die Physiologie der Augenbewegungen, in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 3, p. 536.

55. Guibor, G. P.: Strabismus in Children Corrected by Refraction Alone, Am. J. Ophth. **18**:944, 1935.

56. Keil, F. C.: Summary of Orthoptic Work at the Manhattan Eye, Ear and Throat Hospital, Arch. Ophth. **17**:751 (April) 1937.

57. Thorington, J.: Refraction of the Human Eye, ed. 2, Philadelphia, P. Blakiston's Son & Co., 1930, pp. 278 and 289.

58. Howe, L.: The Muscles of the Eye, New York, G. P. Putnam's Sons, 1907, vol. 2, p. 212.

59. Parinaud,[52] p. 9.

60. Javal,[36] p. 147.

61. Cords,[54] p. 529.

of 2 or 3 drops of a 1 per cent solution of atropine for from three to eight days before refraction, pointing out that partial paralysis of the ciliary muscle leads to increased effort at accommodation and greater convergence when insufficient atropine is used. Both Howe [62] and Thorington [57] emphasized that homatropine is unreliable and should not be used as a cycloplegic for children or young adults having muscle imbalance.

Apparent increase in static hypermetropia shown on successive tests under a cycloplegic is probably to be accounted for by imperfect paralysis of the ciliary muscle on preliminary retinoscopic examinations. Both Brown [63] and Vorisek [64] reported a slight average increase in hypermetropia in children on successive tests. There is generally assumed to be a tendency toward decrease of the normal hypermetropia of children as the eyes develop. When there has been prolonged and careful use of a cycloplegic, it is not usual to find increased hypermetropia on successive tests, though careless administration of drops by parents frequently results in inaccurate measurements, especially for young children.

Changes in refractive errors are to be expected after operations on the muscles. Marshall [65] made a systematic study of such changes and showed that they may occur in eyes not subjected to operation as well as in those operated on.

The question of the proper prescription for the patient with muscle imbalance is one in which clinical experience and theoretic considerations are not always in agreement. In general, convex lenses reduce convergent deviations in which there is an accommodative factor, while concave lenses which increase accommodation for finite distances tend to increase convergent, and reduce divergent, deviations. On the other hand, as reported by Parinaud [66] esotropia in myopic patients sometimes responds to optical correction, probably through a recession of the far point of accommodation and accompanying reduction in convergence; and occasionally divergent deviations in hypermetropic persons are reduced by the use of correcting lenses for the refractive error.

The appearance of the eyes in full cycloplegia is of considerable prognostic value, especially in cases of convergent strabismus. If

62. Howe,[58] vol. 1, p. 169.

63. Brown, E. V. L.: Apparent Increase in Hyperopia up to the Age of Nine Years, Am. J. Ophth. **19**:1106, 1936.

64. Vorisek, E. A.: Changes of the Refraction of Children with Convergent Concomitant Strabismus, Am. J. Ophth. **18**:820, 1935.

65. Marshall, D.: Changes in Refraction Following Operation for Strabismus, Arch. Ophth. **15**:1020 (June) 1936.

66. Parinaud, H.: Le strabisme et son traitement, Paris, Octave Doin, 1899, p. 125.

paralysis of accommodation results in orthophoria or divergence, optical correction can be expected to be effective, provided the refractive error is hypermetropic and convergent deviation is evident when the ciliary muscles are active. Postcycloplegic acceptance by children is completely unreliable as an indication of the proper correction because of active accommodation. Temporary use of atropine is sometimes necessary to reduce spasm of the ciliary muscle when full correction gives blurred vision for distance.

Sattler [67] demonstrated the effectiveness of prolonged occlusion of the fixing eye in improving the acuity of the deviating eye in cases of monolateral strabismus. In 3 of 89 cases amblyopia of the covered eye resulted from this treatment, constituting a strong argument for the existence of amblyopia ex anopsia. Abraham [68] distinguished congenital amblyopia from acquired amblyopia which may result from refractive errors (usually astigmatism of over 2 diopters) or active suppression in cases of strabismus. Smukler [69] suggested that retinal hemorrhages at birth (found in 10 or 15 per cent of the newborn) may cause amblyopia and, subsequently, strabismus. Hesse [70] demonstrated that in a large percentage of cases there are changes in the amblyopic eye which account for its diminished vision. Astigmatism, anisometropia and high hypermetropia are frequently associated with amblyopia. Peter [71] expressed the belief that there may be visible pathologic changes in congenital amblyopia. As pointed out by Parinaud,[72] it is erroneous to assume that improved acuity increases the tendency toward fusion, but because monocular suppression is readily maintained in the presence of unequal acuity, equalization of acuity through accurate optical correction and visual training is usually advocated as preliminary to orthoptic exercise. Complete and constant occlusion of the fixing eye, with frequent and careful tests of acuity (using illiterate charts, even for adults) for both eyes, is accepted as the most effective method of treating amblyopia ex anopsia. For children, such treatment should be delayed until the proper optical correction has been worn for two or three months, since spontaneous recovery of normal acuity may occur. A 4 year old child who showed occasional convergent strabismus was found to have acuity of 6/10 in the right eye and 6/20 in the left eye two

67. Sattler, C. H., cited by Cords,[54] p. 527.

68. Abraham, S. V.: A Classification of Amblyopia, Arch. Ophth. **12**:402 (Sept.) 1934.

69. Smukler, M. E.: Amblyopia and Squint from Head Injuries at Birth, Pennsylvania M. J. **37**:25, 1933.

70. Hesse, R.: Ueber die Amblyopie schielender Augen, Arch. f. Ophth. **130**: 375, 1933.

71. Peter, L. C.: Technic of Orthoptic Training in Squint, Arch. Ophth. **14**:975 (Dec.) 1935.

72. Parinaud,[52] p. 20.

weeks after the prescription of full hypermetropic correction (+ 6.00 sphere for the right eye; + 7.50 sphere for the left eye). There was gradual improvement in acuity, without occlusion; two months later vision was 6/6 in the right eye and 6/10 in the left eye.[73] Pearson and Mayou[74] found that it is difficult to cure amblyopia which has occurred before the second year when treatment is delayed until after the fifth year; Peter[71] expressed the belief that amblyopia does not develop after the seventh year and that the best results in visual training can be expected before the seventh year.

The age, duration of the amblyopia, probable cause for the defect, muscle balance and future treatment should be considered when visual training is recommended by forced use of an amblyopic eye. Young patients respond most successfully (under 6 or 7 years of age); high refractive errors, corrected late in life, result in amblyopia which does not respond well to visual training; amblyopia in conjunction with hyperphoria of 5 diopters or over usually responds poorly to visual training; improvement in acuity is often temporary when binocular vision is precluded by high deviations from normal muscle balance.

Complete occlusion of the fixing eye, which forces central fixation of the defective eye, results in the constant use of central vision in that eye. There is no objective evidence that artificial stimulation by means of mechanical devices is more effective in improving visual acuity than constant use of the eye under ordinary conditions, when continuous stimulation and frequent changes in accommodation are present. If central fixation of the deviating eye is not evident when the fixing eye is covered, occlusion is ineffective in improving acuity. The use of atropine in the fixing eye is effective in transferring fixation to the other eye only if the acuity of the poorer eye is not below 20/50.[75] In cases of severe amblyopia, fixation is maintained by the better eye in spite of the paralyzed accommodation, so that this method can rarely be recommended in the treatment of amblyopia.

Orthoptic training should not be undertaken before the vision of the amblyopic eye has been brought to 20/70 or better. When acuity is not over 20/100, complete macular suppression is easily established, and though fusion of large images is possible, stable binocular vision cannot be stimulated.

Alternate occlusion, suggested by Javal[76] as a method of preventing the establishment of anomalous projection in cases of strabismus, has

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73. Case record 153496 of the Manhattan Eye, Ear and Throat Hospital.

74. Pearson, S., and Mayou, S.: Central London Ophthalmic Hospital: Report of Squint Department, September, 1932-1933, Brit. J. Ophth. **18**:267, 1934.

75. Fuchs,[50] p. 336.

76. Javal,[36] p. 81.

been recommended by some clinicians. Theoretically, if no simultaneous use of the eyes is permitted except during artificial bifoveal stimulation under controlled conditions, anomalous projection might be prevented or discouraged. No extensive clinical studies of this method have been published, but Travers,[77] who used alternate occlusion in 5 cases over a period averaging four and a half months, found it completely ineffective in the treatment of anomalous projection.

Occlusion in cases of strabismus does not change the coordinate movements of the eyes; the covered eye assumes the position ordinarily characteristic of the deviating eye. Prolonged occlusion in cases of heterotropia, which brings about equalization of acuity, may produce diplopia, which is considered an essential to the reestablishment of binocular vision. Actually, such diplopia can usually be observed under test conditions long before acuity is equal in the two eyes, and unless the position of the eyes is such that bifoveal fixation under ordinary conditions (i. e., without prism aids) is possible, observable diplopia is no indication that fusion may be stimulated. The difficulty in orientation caused by constant diplopia brings about prompt return of suppression for most persons. Observant subjects having anomalous projection can sometimes recognize both foveal and peripheral images (monocular diplopia) under test conditions; the images are unequal in clearness.[78]

The following routine is suggested for rapid clinical testing of binocular vision with a major amblyoscope. The eyes should be allowed to recover fully from the effects of cycloplegia before tests are carried out; in practice, though the patient's correction may be placed on the instrument with the use of the proper correcting formulas, preliminary tests should be made with the subject's glasses on. This is particularly true for young patients with hypermetropia who may show spasmodic convergence associated with the stimulation of accommodation resulting from removal of the optical correction. The position of the subject should be relaxed and comfortable, with the instrument adjusted for vision in the primary field, since adduction and convergent deviations are greater in the lower field and abduction and divergent deviations are exaggerated in the upper field. It is unnecessary to hold the head in position with the hand except when marked head tilt or severe amblyopia is present. An intelligent child learns to report not what he actually sees but what he believes he should see, so that extreme care is necessary to avoid suggestion which may falsify tests. Control of illumination makes accurate checking of the responses possible.

1. The instrument is adjusted to the proper height and interpupillary distance.

77. Travers,[28] p. 71.
78. Travers,[28] p. 69.

2. Slides having small, centrally placed, dissimilar images are inserted, and the following tests are carried out:

(*a*) Monocular fixation is tested by presenting the images separately to each eye, noting the angle gamma (or alpha) as positive or negative. (In normal and hypermetropic eyes the angle between the optic axis and the visual axis at the nodal point of the eye is usually positive, the light reflex lying to the nasal side of the apex of the cornea and giving rise to apparent divergence; in myopic eyes the angle gamma may be zero or negative, in which case the light reflex lies temporal to the corneal apex and gives apparent convergence. With appropriate slides, the size of the angle may be estimated in degrees.)

(*b*) On testing monocular motility any limitation of motion in the lateral fields is noted.

(*c*) The objective deviation is measured by alternate presentation of the images, prismatic correction being added by adjusting the images to neutralize lateral and vertical movements. (In cases of concomitant strabismus the primary deviation, measured with the fixing eye in the primary position, is equal to the secondary deviation, measured with the deviating eye in the primary position.) If foveal fixation is not present, objective measurements must be made by adjusting the corneal reflexes.

(*d*) The subjective deviation is measured by noting the prismatic correction necessary for superposition of small, unlike images. (When there is normal projection, subjective and objective measurements are equivalent; extinguishing either image after subjective superposition does not result in movement of the opposite eye. When there is anomalous projection, the subjective deviation is less than the objective deviation. Paradoxic diplopia is reported with the prism which corrects the deviation objectively; superposition is attempted by decreasing the prismatic correction, which makes the images appear to approach. Actual superposition is frequently impossible, one image disappearing only to reappear on the opposite side of the other image. The presence of anomalous projection is substantiated, first, if one image is reported to be less clear than when it is seen monocularly; second, if the corneal reflex of one eye is seen to lie in a peripheral position not compatible with central fixation; and third, if repeated tests indicate unstable binocular vision, with variability in the prismatic correction required for superposition.)

3. Slides having similar images, with cues to indicate binocular fixation and fusion, are inserted, and the following tests are carried out:

(*a*) Abduction is tested by adding prisms base in or reducing prisms base out until diplopia is reported or until one image is suppressed. (Abduction measured in this way, on a major amblyoscope, is usually higher than that measured by loose prism methods.)

(*b*) Adduction is tested by adding prisms base out or reducing prisms base in until diplopia is reported or until one image is suppressed. (When subjective reports are unreliable, amplitude of fusion can be estimated by noting the points at which binocular fixation is replaced by alternation or monocular fixation.) Convergence micropsia, the apparent decrease in the size of the fused images as prisms base out are added, is frequently reported spontaneously and is a supplementary indication of fusion, since when binocular fixation is lost the images appear larger.[79]

(*c*) Supraduction or subduction is tested by adding prisms base up or base down; cycloduction measurements probably have no validity, as it has been shown by Beasley and Peckham that torsional movements are not made under such conditions.[80]

4. Slides for testing stereopsis (disparate images) are inserted next.

(*a*) Gross stereopsis is tested by ascertaining the depth relation apparent on slides having high binocular disparity, similar parts of the images being widely separated when the slides are held together. Stereopsis is defective if disparate parts of the images appear displaced to one side (i. e., if a ball which should be centered in a ring is displaced to one side). Such displacement is usually observed when the acuity of one eye is much better than that of the other eye.

(*b*) Fine stereopsis is tested by using graded slides with progressive reduction in the disparity of the paired images.

The same tests may be repeated for the near point by adding appropriate concave lenses to stimulate accommodation or by moving the images closer to the eyes. Since the optical effect of concave lenses decreases when they are moved from the eyes, the lenses employed must be selected accordingly. With accommodation for the reading distance (33 cm.), the person with an interpupillary distance of 60 mm. must converge about 18 prism diopters. Prism tolerance for near vision should be measured from this point and recorded as prism tolerance for base in or base out with accommodation.

An attempt to define "normal" ductions is complicated by the high individual variations among those having no symptoms of muscle imbalance and by the fact that measurements of duction power vary with age, fatigue, practice and psychologic factors. Moreover, the method employed influences the absolute measurements. The ratio of adduction to abduction has been regarded as of greater clinical significance than the absolute values of positive or negative convergence. It is usually stated that adduction with distance fixation should be from two to four

79. Travers,[28] p. 66.

80. Beasley, W. C., and Peckham, R. H.: An Objective Study of "Cyclotorsion," Psychol. Bull. **33**:741, 1936.

times abduction to assure comfortable convergence reserve.[81] Howe, pointing out that interrupted increases in prismatic addition give lower measurements than continuous increases, recommended what he called the minimum method of measuring ductions [82] by beginning with a prism too great to be overcome. This method was officially adopted by the American Medical Association in 1907, but is seldom employed.[83] Maximum adduction cannot be accurately determined because it is easily increased by practice. The published statistical studies of average duction power show great variability. In 1927 Berens, Losey and Hardy [24] published average values for adduction and abduction for distance and near fixation for 218 young men having normal acuity and no ocular symptoms and for 104 patients selected as having normal muscle balance. All were measured by loose prisms, presented in racks, from low to high.

Distance Fixation	Near Fixation
Adduction: 9 to 45D.; average, 19D.	Adduction: 15 to 70D.; average, 41D.
Abduction: 5 to 15D.; average, 7D.	Abduction: 14 to 38D.; average, 19D.

For rapid indication of fusion at distance or at the near point [84] Worth's four dot test is a convenient supplementary method.[85] Four illuminated circles, a white and a red circle and two green circles, are observed through red and green complementary filters: A subjective report of four lights indicates fusion; two or three, suppression; and five, diplopia. (If this test is prepared by the examiner, care must be used to secure strictly complementary filters, without which the test is invalid.)

The clinical training of binocular coordination implies the stimulation of normal projection, the training of adequate fusion amplitude and the encouragement of stereopsis.

It has been assumed that anomalous projection, in which bimacular stimulation gives diplopia, is an acquired perceptual system and that normal projection can be reestablished by the use of appropriate orthoptic methods. This technic has been described in detail by Pugh,[86] Lyle and Jackson [87] and Travers.[88] To review briefly, two methods may be described:

1. Simultaneous stimulation of the two macular regions at the objective angle of deviation, with repeated lateral movements of the image.

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81. Taylor, E. A.: Controlled Reading, Chicago, Chicago University Press, 1937, p. 246 (footnote).

82. Howe,[58] vol 1, p. 299.

83. Wells,[37] p. 25.

84. Worth Dot test (modified for near fixation), Clairmont Nichols Company, New York.

85. Lyle and Jackson,[21] p. 37.

86. Pugh,[41] p. 61.

87. Lyle and Jackson,[21] p. 58.

88. Travers,[28] p. 68.

2. Alternate presentation of images at the objective angle of deviation by manual or mechanical control of illumination.

The infrequent success of orthoptic treatment in the correction of anomalous projection may be accounted for by the fact that even prolonged bimacular stimulation under highly artificial conditions cannot bring about a stable projection system unless the eyes are in a position which makes constant bifoveal fixation possible. Travers [89] found that successful treatment of anomalous projection by orthoptic methods is so rare that it cannot be legitimately advocated in any case, since no treatment which did not include operation had any beneficial effect on patients with this type of projection. Lyle and Jackson [90] are more optimistic with regard to the use of orthoptic training in such cases, stating that in a favorable case projection becomes "true" in twelve training periods. Nevertheless, in their records of 24 cases of monolateral convergent strabismus in which the treatment did not include surgical measures, an average of eighteen periods was required to obtain "normal correspondence," and only 50 per cent of the patients showed single binocular vision at the end of training (after six to ninety periods). Pugh [91] did not state how long training should be continued in cases of anomalous projection, but she pointed out that the first method, mentioned previously, "takes a long time to obtain a favorable result, too long for the perseverance of the average patient." In regard to the second method, with alternate presentation of images at the rate of from one to twenty per second, Pugh stated that the patient "soon accepts the images binocularly in their correct relationship and sees one composite image all the time." Nevertheless, in evaluating the results of treatment by orthoptic training, Pugh excluded all cases of anomalous projection (50 per cent of the cases).[92]

Authorities are not in agreement concerning the value of orthoptic training in conjunction with surgical treatment. Unfortunately, it is not possible to isolate the specific contribution of orthoptic training toward the return of binocular vision in such cases, since binocular vision may be reestablished spontaneously after operation. Bressler [93] reported more favorable results in a group of 36 cases in which both preoperative and postoperative orthoptic training was used than in 150 cases in which operation alone was employed. His results, given in percentages, may be misleading because of the fact that the groups compared are not

89. Travers,[28] p. 77.

90. Lyle and Jackson,[21] p. 97.

91. Pugh,[41] pp. 62 and 85.

92. Pugh,[41] p. 85.

93. Bressler, J. L.: Treatment of Strabismus: Influence of Orthoptic Training on the Results of Operation, Arch. Ophth. **16**:433 (Sept.) 1936.

equivalent. Berens, Payne and Kern,[94] in a study of orthoptic training and surgical treatment in cases of hyperphoria and hypertropia associated with lateral deviations, showed that prolonged periods of preoperative and postoperative training, possible for patients in private practice, apparently contribute to more satisfactory binocular vision. They are of the opinion that anomalous projection, uncorrected preoperatively, may cause the postoperative recurrence of the same deviation. They reported, however, that placing the eyes in approximate parallelism by means of surgical methods resulted in an increase of 40 per cent in the number of patients who could fuse.[94] Hicks and Hosford[95] reported spontaneous postoperative fusion in 18 of 24 cases in which no orthoptic training was used. Travers[96] stated: "The idea that an abnormal correspondence will forcibly deviate the eyes after operation into their old position is also probably without justification."

If normal projection is present and fusion can be elicited on binocular instruments with or without prismatic aids, increased amplitude of fusion through training of adduction and abduction may be brought about by practice. Paired similar images with binocular cues should be used, and the patient must be taught to observe diplopia or suppression when binocular fixation is lost. It has been assumed, without adequate theoretic basis or controlled research, that practice in adduction will lessen exophoric deviations, while practice in abduction will lessen esophoric deviations. It is well established that voluntary control of adduction can be assisted by exercises with base-out prisms, and symptomatic relief in convergence insufficiency may be reported when adduction reserve is high, as indicated by Lyle and Jackson,[97] Berens[24] and Stutterheim.[98] Berens suggested that tolerance to a base-out prism of 75 prism diopters for both distance and the near point should be attained by home exercise with loose prisms. Such exercise often causes esophoria, which, according to Berens,[24] does not produce symptoms. In divergence excess (in which the deviation on dissociation tests is greater for distance than for the near point), adduction, both at the far and at the near point, can sometimes be stimulated, and voluntary control of binocular fixation can occasionally be established, but the deviation will remain unchanged with dissociation tests or when the

94. Berens, C.; Payne, B. F., and Kern, D.: Orthoptic Training and Surgery in Hyperphoria and Hypertropia Combined with Lateral Deviations, Am. J. Ophth. **18**:522, 1935.

95. Hicks, A. M., and Hosford, G. N.: Orthoptic Treatment of Squint, Arch. Ophth. **13**:1026 (June) 1935.

96. Travers,[28] p. 86.

97. Lyle and Jackson,[21] p. 153.

98. Stutterheim, N. A.: Eyestrain and Convergence, London, H. K. Lewis & Co., Inc., 1937.

eyes are at rest. In such cases it may be necessary to make use of accommodative convergence by beginning adduction at the near point; in high divergence excess it is not always possible to establish any binocular vision for distance. Since the normal tendency toward divergent position of the eyes during growth prevents the spontaneous cure of divergence excess, early operation is sometimes to be recommended to prevent a cosmetic defect and to avoid the additional convergence insufficiency which may occur. Abduction, or tolerance to base-in prisms, is involuntary and must be stimulated reflexly through the relaxation of convergence. Javal [99] expressed the belief that strong adduction under voluntary control must be learned before abduction can be accomplished through the relaxation of convergence. Abduction is usually undertaken with accommodation relaxed (i. e., with distance fixation), but Pugh [100] reported more favorable results when accommodation, without convergence, is stimulated by placing the images close to the eyes, without additional base-out prisms (possible on the orthoptoscope or the stereoscope). Tait,[101] who used controlled exercise with base-in prisms for 4 normal subjects, demonstrated that such exercise served to increase adduction power and to decrease exophoria. It is evident that since fatigue and other factors may be of considerable significance in duction training, further research rather than haphazard clinical work should be undertaken.

Careful tests of muscle balance may disclose esophoria for distance and exophoria for the near point or, particularly in cases of postoperative convergent concomitant strabismus, exophoria for distance and esophoria for the near point. It is probable that in such cases duction training should include practice with base-in and base-out prisms. Temporary esophoria for distance is frequent after extensive close work, even when there is marked exophoria at the near point. Persons who show exophoria at the near point, combined with esophoria for distance and low adduction, may overcompensate for a primary exophoric deviation, and both Wells [102] and Lyle and Jackson [103] advocate exercise with base-out prisms for such persons.

Within narrow limits, it may be possible to train a hypermetropic person to accommodate without excessive convergence, but such exercise does not usually result in any permanent benefit, since persons predisposed to convergent deviations cannot accommodate sufficiently

99. Javal,[36] p. 111.

100. Pugh,[41] p. 54.

101. Tait, W. J.: Further Studies of the Fusional Convergence Amplitude, Am. J. Optometry **14**:4, 1937.

102. Wells,[37] p. 70.

103. Lyle and Jackson,[21] p. 152.

for clear vision without stimulation of convergence. It is obvious that young hypermetropic persons whose eyes are orthophoric with correaction, with stable fusion at both the near point and at distance, should not be subjected to prolonged orthoptic training in an effort to establish the same coordination without correction of the refractive error. Parents are sometimes under the impression that exercise will make possible the discarding of children's glasses, but since this is rarely indicated, they should be correctly informed.

There is little basis for the assumption that stereopsis can be "taught" by orthoptic methods. Moreover, it is erroneous to assume that stereoscopic perception has a direct influence on muscle balance. Patients, for instance, who show periodic exotropia may demonstrate perfect stereoscopic perception under test conditions, and yet they do not employ binocular vision at all times. In fact, it is doubtful if even normal persons make use of stereoscopic vision constantly. Unless the conditions requisite to stereopsis are fulfilled—approximately equal acuity in the two eyes, bifoveal fixation, etc.—stereoscopic depth cannot be observed. Stereoscopic charts may be used in training ductions, and the subject may be encouraged to observe the depth relation mediated by binocular disparity, but "mental effort" cannot establish stereopsis.

Postoperative duction training does not differ essentially from preoperative training and can be undertaken as soon as healing has occurred. Passive tests of binocular vision and ocular position may be carried out shortly after operation, care being taken not to disturb the stitches by extreme movements. Changes in optical correction are frequently indicated postoperatively, and the influence of the correction on ocular position may be measured on a major amblyoscope. Trial lenses should be worn fifteen minutes before such tests.

Home exercise as a supplement to clinical training is frequently advocated, but most authorities do not report successful results, since even with competent instruction the exercises are seldom carried out regularly. It should be emphasized that home exercises based on prism technic or the use of a stereoscope can be attempted only when fusion with normal projection can be elicited on clinical tests. Accurate preliminary diagnosis of binocular vision is therefore necessary. Explicit directions, which must be modified to suit the individual patient, with an explanation of the purpose of the exercises, should be given. The following outline suggests instructions for simple home exercises:

1. Simple convergence exercises to stimulate convergence with accommodation, indicated in convergence insufficiency or in divergence excess to prevent secondary convergence insufficiency. (These exercises are not indicated if convergence cannot be elicited on repeated

trials.) Convergence exercises are used to help sustain normal coordination when one eye has a tendency to turn out. The instructions follow:

Use a black vertical line, a vertical row of letters or, for children, small toys or tiny pictures. Hold the object at arm's length, slightly below the level of the eyes; watch it closely with both eyes as it is moved slowly toward the nose. When the object appears double, or when one eye moves away from the nose, repeat the test, starting with the object at arm's length. Be sure that both eyes are focused on the object. (They should be seen to move toward the nose.) Move the object slowly. Repeat————times————periods daily.

(For children, ten repetitions three times daily may be recommended; for adults, three five minute periods. Presbyopic patients seldom attain any symptomatic relief from such exercise.)

2. Prism convergence exercises to stimulate convergence, with or without accommodation, indicated in convergence insufficiency, divergence excess or esophoria if adduction is low. The instructions follow:

Obtain prisms no.————and a red glass filter from an optician for temporary use. Arrange a light (15 watt frosted bulb, flash light or candle) which can be observed from a distance of 20 feet (609 cm.) Place a prism base out (the thick side toward the temple) and a red glass before either eye. If two lights are seen, make an effort to bring the lights together by turning the eyes toward the nose (possible by looking momentarily toward a near object with the prism in position). After fusion is obtained, so that a single light is seen through the prism, count five slowly, remove the prism and repeat. Practice————minutes ————times daily. Increase to no.————prism.

(Three five minute periods daily may be recommended. Exercise should begin with the highest prism easily overcome on clinical tests and increased weekly until 40 diopters or more can be overcome. If the use of a red glass prevents fusion, tests may be carried out without the filter: Monocular fixation is evident when a cover placed quickly before the fixing eye causes inward movement of the opposite eye.)

3. Stereoscopic duction exercises to increase fusion amplitudes and to dissociate accommodation and convergence, indicated in exophoria or esophoria when fusion can be elicited with normal projection and without high prism correction. The instructions for these exercises follow:

Obtain from an optician a stand stereoscope and set of divided stereoscopic charts.

Adduction A. (*a*) Adjust the stereoscope for distance, placing the lenses ————inches from the chart holder.

(*b*) Find the position in which fusion is attained by superposition of unlike images (pictures in which the two images have no similar parts).

(*c*) Substitute similar paired charts, placing them in the position found in 2; be sure the images are fused by looking for all the objects in the picture.

(*d*) Move one or both charts toward the center of the chart holder until two pictures are seen or until some part of the fused picture disappears. Keep a record to show how close together the charts can be moved without losing fusion. Practice from ten to fifteen minutes daily.

Adduction B. (*a*) Adjust the stereoscope for near fixation by placing the lenses————inches from the chart holder.

(*b*) Fuse the divided charts, leaving them as close together as possible, then move the stereoscope from the cards until fusion is lost. Practice from five to ten minutes daily.

Abduction A. Instructions (*a*, *b* and *c* are the same as given for Adduction A).

(*d*) Move one or both charts away from the center of the chart holder until two pictures are seen or until some object in the fused picture disappears. Practice from ten to fifteen minutes daily.

Abduction B. (*a*) Adjust the stereoscope for distance by placing the lenses ————inches from the chart holder.

(*b*) Fuse the divided charts, leaving them as far apart as possible; then move the stereoscope closer to the charts until fusion is lost. Practice from five to ten minutes daily.

The foregoing exercises are simplified if a stereoscope having a calibrated shaft is used. Otherwise, the positions for distance and for near fixation must be calculated by finding the first focal plane ("optical infinity"), which is determined by the strength of the decentered convex spheres in a lenticular stereoscope.

Intelligent cooperation on the part of those responsible for the home care of strabismic children is essential and can be secured by giving information to the parents concerning the purpose and sequence of therapeutic measures. Parents are chiefly concerned with the child's appearance, and it is necessary to emphasize that restoration of binocular vision may contribute to the maintenance of normal ocular position. In clinical work at the Manhattan Eye, Ear and Throat Hospital, a booklet, "Helping Children See Straight," [104] prepared to answer parents' questions, has been found useful.

SUMMARY

Binocular vision, of relatively recent development in the evolutionary scale, is based on complex neurologic relations which are not completely understood. Although the essentials for binocular vision can be isolated, the exact basis for the production and maintenance of fusion remains in the realm of theory. Clinical analysis of the anomalies of binocular vision which are associated with imbalance of the ocular muscles frequently reveals significant functional changes, including defects of the visual field (suppression scotomas), amblyopia ex anopsia and anoma-

104. Shaad, D. J.: Helping Children See Straight: For Parents, New York, The Manhattan Eye, Ear and Throat Hospital, 1937.

lous projection. Orthoptic procedure implies the application of measures designed to correct these defects and to promote normal coordination of the ocular muscles through the training of fusional ductions. If such treatment is to be established as a routine therapeutic measure in the treatment of strabismus, the value of its contribution depends on the proper testing of binocular vision, adequate records and careful control of other factors, such as correction of refractive errors, which form a part of the general treatment. Since clinical studies are frequently incomplete and confined to selected cases, the role of orthoptic methods in the restoration of normal binocular vision cannot be accurately outlined without further research.

Correspondence

SIMPLIFICATION OF THE O'CONNOR CINCH OPERATION

To the Editor:—I read with deep interest in the August issue of the ARCHIVES, page 315, Dr. O'Connor's comment on my article entitled "Simplification of the O'Connor Cinch Operation," published in the June issue.

Dr. O'Connor stated that I have "fallen into the common error" that he (Dr. O'Connor) always divides the tendon into four strips. I know that he does not always divide the tendon into four strips. However, I invariably do for the type of case mentioned in my article, and for the following reasons:

1. To remove the danger of tearing a strip of tendon. When the tendon is divided into more than four strips—Dr. O'Connor even suggests up to twelve strips in some cases—one of these delicate thin strips (often 1 mm. or less wide) is occasionally torn when the strip is pulled aside for looping, no matter how gently the narrow strip is handled. This is most likely to happen in operations on children, the muscle being often attenuated or the field of operation limited. Other surgeons have called attention to this danger. (Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936, p. 1150).

2. As Dr. O'Connor has himself stated, "Width of tendon strip. The more tissue in each, the greater the shortening. . . ." (The Cinch Shortening Loop in Surgery of the Extra-Ocular Muscles, *West. J. Surg.* **39:** 670 [Sept.] 1931).

If Dr. O'Connor had read my article carefully, he would have noted that I made not the slightest reference to the use of the separators or to the number of strips into which the tendon is to be divided in the correction of vertical deviations.

I stated that it was my practice in all cases of divergent squint and properly selected cases of convergent squint of moderate degree to divide the tendon and muscle into three or four strips, using the separators. The advantages of this procedure are as follows:

1. A good view of the operative field is presented.

2. Less manipulation and less risk of tearing the strips are involved, as the strips do not have to be pulled aside to loop them.

3. The operative procedure is greatly facilitated and shortened.

The results have been uniformly gratifying in the type of case in which I suggested that my procedure be used.

M. E. SMUKLER, M.D., Philadelphia.

A NEW MODEL GONIOSCOPE

To the Editor:—In the report on a new model gonioscope in the June 1938 issue of the ARCHIVES (page 983) no mention was made of the portable gonioscope previously used and exhibited by Dr. Otto Barkan. No reference was made to his article in the December 1937

issue of the *American Journal of Ophthalmology* (page 1237) for the reason that the article by Mr. Goebel and me was written before that date. The fact that he had exhibited his instrument before the American Academy of Ophthalmology and Otolaryngology and elsewhere and his paper in the 1936 *Transactions of the Academy of Ophthalmology and Otolaryngology* were inadvertently overlooked. The stand described in our paper is similar to Dr. Barkan's, and he properly deserves full credit for it. However, I believe that the main features of our instrument, namely, the lighting arrangement, inverted suspension to allow free examination of the angle in its entire circumference and centered pivot to keep the instrument in focus as various segments of the angle are explored, are both new and definite improvements. There was no intention of taking any credit for the stand away from Dr. Barkan, and I hope that this letter will correct any such impression that may have been given.

JOHN M. MCLEAN, M.D., Baltimore.

News and Notes

PERSONAL

Notice has been received of the death of Mr. Jujiro Komoto, honorable professor of the Tokyo Imperial University. Professor Komoto was born on Aug. 16, 1859, in Hyogo-ken. After graduation from the Tokyo Foreign Languages School and the Department of Medicine of Tokyo University in 1883, he was sent abroad by his government, where he continued his studies in Germany and Austria from 1885 to 1889. On his return from study abroad he was appointed professor of the College of Medicine of Imperial University, Tokyo, in June 1889 and was simultaneously appointed to the chair of ophthalmology in the same college. The former College of Medicine became the Faculty of Medicine in 1915, and Professor Komoto was appointed (in succession) again as professor of ophthalmology. Almost all of his important publications were in Japanese. He died on April 4, 1938, at the age of 79, after a long and painful illness.

SOCIETY NEWS

Journal of Social Ophthalmology.—The International Association for the Prevention of Blindness, is publishing a new organ, the *Journal of Social Ophthalmology,* for the purpose of keeping before the minds of social workers and health officials the importance of the health of the eye and the economic disability that arises from blindness. It is not a clinical journal. The first number is printed in English and French in parallel columns. As occasion suggests, other languages may be employed. The journal is sent free to subscribers to the association, 66, Boulevard Saint-Michel, Paris.

GENERAL NEWS

Exhibition at the Royal Eye Hospital.—An exhibition dealing with the prevention of industrial injuries to the eyes was opened at the Royal Eye Hospital (Royal South London Ophthalmic Hospital, Inc.) on June 22 by the Earl of Athlone, K.G. Addresses were made at the opening of the exhibition by the chairman of the hospital, Mr. L. Vernon Cargill, F.R.C.S., the Earl of Athlone and Mr. J. Minton, F.R.C.S., medical representative of the Industrial Eye Injuries Committee.

The exhibition included a display of different types of goggles for such occupations as grinding, welding and riveting and metal-sputtered goggles, glare and dust proof, which are used by pilots of the Royal Air Force and Imperial Airways. A film was shown at the opening of the exhibition illustrating the protection of eyes in industry. It showed a workman suffering from an ocular injury received because he was not wearing his goggles or using a safety screen. Men working in factories doing various types of engineering work were shown using the necessary safety appliances.

Other demonstrations included one of oxyacetylene welding and electric welding by workmen wearing goggles and protective screens. Large photographs around the walls showed other workers either using or neglecting to use protective appliances.

A committee has been formed by the hospital to further the object of making employers conscious of the necessity of safety measures, and, a matter of perhaps equal importance, of combating prejudice or negligence among workmen whom the appliances are intended to protect. Part of the exhibition is to remain in the casualty department of the hospital as a permanent museum. It will thus come to the notice of the 7,000 workmen who are treated at the hospital every year for ocular injuries, many of them serious.

CORRECTION

In the abstract of the report by Dr. I. S. Tassman entitled "The Use of Paredrine in Cycloplegia," which appeared in the proceedings of the College of Physicians of Philadelphia, Section on Ophthalmology for March 17, 1938, in the July issue of the ARCHIVES (**20**:156, 1938), the second sentence in the fifth paragraph should read:

"After experimenting with solutions of different strengths, it was found that a 4 per cent solution of homatropine hydrobromide followed in three minutes by 1 drop of a 1 per cent solution of paredrine hydrobromide produced satisfactory results."

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Congenital Anomalies

COLOBOMATA OF THE OPTIC NERVE SHEATH IN RATS. J. V. V. NICHOLLS and K. TANSLEY, Brit. J. Ophth. **22:** 165 (March) 1938.

This study is based on the results of examination of histologic sections of the eyes of 10 rats. There were ectasia of the retina passing between the edge of the lamina cribrosa and the sclera into the optic nerve sheath. The essential feature in this series was poor development of the lamina cribrosa. There is some difficulty in evaluating the relation of the ectasia to the weakness of the lamina cribrosa. Whether the ectasia follows the collapse of the lamina or both conditions are part of the same process is difficult to determine. The second view is supported by Kayanaga. According to him, there is an abnormality in the condensation of the mesoderm about the neck of the primary optic vesicle, so that small pouches of neural ectoderm are nipped off about the future nerve head. Because of, or along with this, there is poor development of the lamina cribrosa. This explains how in some of the authors' cases the ectasia completely surrounded the optic nerve within its sheath, a previously undescribed condition.

W. ZENTMAYER.

Experimental Pathology

THE GALACTOSE CATARACT: CLINICAL OBSERVATIONS. T. SASAKI, Arch. f. Ophth. **138:** 351 (Feb.) 1938.

Sasaki made thorough clinical (including use of the slit lamp) studies of the ocular changes which occurred in young rats kept on a diet consisting of 50 per cent of galactose. The observations of Mitchell, Dodge, Yudkin and Arnold with regard to galactose cataract were confirmed. The galactose cataracts were found to have a characteristic clinical picture and to take a regular course. The first opacities developed in from three to seven days after the diet was started; these opacities were situated around the sutures and were subsequently covered by subcapsular radial (spokes and water clefts) opacities. The cataracts became complete within from twenty-two to twenty-eight days.

P. C. KRONFELD.

THE PROPERTY OF GALACTOSE CATARACTS TO PROTECT VITAMIN C FROM OXIDATION. T. SASAKI, Arch. f. Ophth. **138:** 380 (Feb.) 1938.

The cataractous lenses of rats fed a diet consisting of 50 per cent of galactose protect (in in vitro experiments) vitamin C from oxidation. This property of galactose may be due to the diffusion of a reducing substance (glutathione?) out of the lens into the surrounding solution,

which contains vitamin C. The increased permeability of the capsule of a galactose cataract would facilitate this diffusion.

P. C. KRONFELD.

General Diseases

OCULAR LEPROSY: REPORT OF TWENTY-TWO CASES. VILLARD, BOUNIOL, VIALLEFONT and FRIENTÈS, Bull. Soc. d'opht. de Paris, July 1937, p. 348.

Of 31 lepers examined in a colony at Valbonne, 22 presented ocular lesions. A short résumé of the ocular changes noted in each patient is given. The authors comment on the frequency and the gravity of ocular complications in cases of leprosy; on the fact that so many of the patients had contracted leprosy in France, and on the uncharacteristic nature of the ocular lesions as compared to those associated with tuberculosis. Except for a type of iritis with small white grains formed on the collarette of the iris, no characteristic sign of leprosy was noted. Scleral and episcleral lesions were not abundant in this group. Operative procedures were seldom used in the treatment of ocular leprosy, but of those necessary, iridectomy was most often required. The authors feel that complications from leprosy and tuberculosis are comparable in relation to involvement of the eyes.

L. L. MAYER.

General Pathology

FURTHER CONTRIBUTION TO THE KNOWLEDGE OF GUNN'S SIGN. L. SALLMANN, Arch. f. Ophth. 138: 412 (Feb.) 1938.

A man aged 77 suffered from a decompensated hypertensive cardiovascular disease with arteriosclerosis and arteriolosclerosis. Ophthalmoscopic examination revealed narrow and straight retinal arteries with exaggerated reflex stripes and delicate white sheaths. Nicking of the veins was present at several places. Pathologic examination of these places revealed as cause of the nicking a definite thickening of the adventitia of both vessels at the place of the crossing. Nicking of the veins, or Gunn's sign, is, therefore, a sign of localized vascular sclerosis and not of displacement of the vein by the rigid artery.

P. C. KRONFELD.

Lens

ARREST OF NUTRITIONAL CATARACT BY USE OF RIBOFLAVIN. P. L. DAY, W. J. DARBY and K. W. COSGROVE, J. Nutrition 15: 83 (Jan.) 1938.

Day and his associates gave young albino rats a diet deficient in riboflavin and examined their eyes at frequent intervals with the ophthalmoscope. Of 16 control animals receiving the deficient diet without supplement, cataract developed in 13 in an average of fifty-two days. The cataract proceeded to maturity in 12 of these rats in an average of sixty-seven days. The average time of survival was seventy-four days. When early cataractous changes were evident, 25 rats were given intra-

muscular injections of riboflavin in doses of 120 micrograms twice a week. The animals rapidly increased in weight; new hair appeared on those rats with alopecia, and keratitis cleared up slowly. In 11 of these rats cataract was arrested in both eyes. In each of 6 other rats the cataract proceeded to maturity in one eye, but its progress was definitely arrested in the other eye. The cataract proceeded to maturity in both eyes in 2 rats. Six rats were found to have clear lenses in both eyes after the keratitis cleared. It is thus apparent that the progress of the development of cataract was arrested by the administration of riboflavin in 17 of 19 animals exhibiting cataract. These data furnished additional evidence that riboflavin is the cataract-preventive vitamin.

J. A. M. A. (W. ZENTMAYER).

EXFOLIATION OF THE LENS CAPSULE IN GLAUCOMA. A. GARROW, Brit. J. Ophth. **22:** 214 (April) 1938.

Garrow gives a brief review of the literature on exfoliation of the capsule of the lens in cases of glaucoma, describes the several types of exfoliation which have been observed and gives a detailed description of his own cases with line drawings of the appearance in each case.

He gives the following summary:

Fifty-one glaucomatous patients were examined for exfoliation of the capsule. This condition was found in 8. Two of these patients had a congestive attack when they first came under observation.

No record was kept of the number of nonglaucomatous patients examined with the slit lamp during this investigation, but exfoliation of the capsule was found only twice in such patients.

The article contains several plates.

W. ZENTMAYER.

TECHNIC OF TOTAL EXTRACTION OF CATARACT WITH FORCEPS. R. DE SAINT-MARTIN, Ann. d'ocul. **174:** 750 (Nov.) 1937.

The April 1937 issue of the *Annales d'oculistique* contains an interesting article by Dr. Porte, of Geneva, on the intracapsular extraction of cataract. In this article he disapproves of the technic of extraction with forceps, which today is considered classic and is known under the names of the technic of Stanculeanu, Knapp, Török and Elschnig. Elschnig renamed this technic the "Hungarian method."

The author of the present article accepts the superiority of this method without discussion. He gives his reasons why he considers the conclusions of Dr. Porte unreliable and liable to mislead operators who are not too familiar with surgical work on the lens and consequently should not undertake this type of operation.

S. H. MCKEE.

Lids

STAPHYLOCOCCIC SEPTICEMIA FOLLOWING A SUPPURATING MEIBOMIAN CYST: REPORT OF A CASE. R. GOZBERK, Ann. d'ocul. **175:** 159 (Feb.) 1938.

A furuncle or anthrax at any site may be the cause of staphylococcic septicemia. In this respect furuncle of the face merits special consider-

ation, as anastomosis of the facial vein with the superior ophthalmic vein renders the passage of micro-organisms toward the cavernous sinus easy. Suppurative phlebitis of the orbital veins and of the cavernous sinus is a not uncommon complication of furuncle of the side of the nose, the eyebrow, the forehead or the upper lip.

Different authors are cited as having published reports of cases from 1871 to 1933. A case is reported in detail. A boy of 11 years was seen because of an acute inflammatory exophthalmos, with most serious symptoms. The child had had measles the previous month, and convalescence had been interrupted by acute enteritis. One week before the child was seen, there appeared on the right upper lid a reddish swelling, and three days later chills and high fever suddenly developed; in two days a marked proptosis of the eye occurred. There followed a generalized staphylococcic septicemia, with abscesses in the orbit and conjunctiva. Postmortem examination showed abscesses in the brain, lungs and kidneys, all of which had the same character. It is believed that the measles lowered the resistance of the patient to such an extent that secondary infection progressed easily. Two illustrations accompany the article.

S. H. McKee.

Methods of Examination

A Simple Accessory for the Dark Room. J. Sherne, Brit. J. Ophth. 22: 109, 1938.

The device described here is a reflector such as cyclists use instead of a rear light. It is affixed to the wall of the darkroom directly opposite to and on the same level as the retinoscopic light. It produces a brilliant red fixation spot; this interests children and can be seen by even the most myopic. It is a great advance over the other methods for the production of relaxation of accommodation.

W. Zentmayer.

The Use of Sodium and Mercury Vapor Lamps in Ophthalmology. A. J. Ballantyne, Brit. J. Ophth. 22: 204 (April) 1938.

Ballantyne describes a convenient form of mercury vapor lamp of durable construction and of moderate cost. For most practical purposes, it is a satisfactory substitute for the Vogt red-free lamp, and it has the advantage that it is less disagreeable for the patient and creates less dazzle for the examiner.

The sodium vapor lamp has the important qualities that it is almost purely monochromatic and that the light is largely absorbed by the blood in the retinal vessels. These two properties give the light a notable resolving power.

The author describes the details of the fundus as observed under the two types of lamp.

W. Zentmayer.

Neurology

Neural Correlations of Vision and Their Significance for Localization of Tumors of the Brain. C. A. Elsberg and H. Spotnitz, Arch. Neurol. & Psychiat. 39: 315 (Feb.) 1938.

The authors present a preliminary report in which they describe functional visual tests, such as dark adaptation, flicker and the threshold

for light as related to minimal lighting and the area of illumination. They attempt to interrelate these visual functions in the normal person and in the person with a pathologic process of the visual tracts. Using white objects against a black background and white test objects with a black center, they found varying relations between the area of the test object and the minimum light required for vision, which, for each person, could be formulated into constants. This raised the question as to whether or not the constants were dependent on the amounts of energy received at the cones, bipolar cells and ganglion cells of the retina. The authors felt that the interrelations found did demonstrate that summation and inhibitory effects are important in retinal perception.

The use of these functional visual tests for the localization of lesions in the retina and visual tracts is discussed and illustrated. The authors realize that the study is as yet of theoretic interest only, but they are continuing the work, as there are indications of practical possibilities. Ophthalmologists should be interested in this article from the standpoint of visual physiology.

R. IRVINE.

A CASE OF INTRAOCULAR NEUROMA (RECKLINGHAUSEN'S DISEASE) OF THE LEFT OPTIC NERVE HEAD. H. B. STALLARD, Brit. J. Ophth. **22**: 11 (Jan.) 1938.

Stallard enumerates and describes in some detail the ocular complications of Recklinghausen's disease.

The author gives the following résumé of the case reported: A young man aged 19 years had a neuroma affecting the optic nerve head of the left eye. His family history showed that in the two generations preceding his there had been 2 cases of multiple neurofibroma affecting the central nervous system and the nerves serving the special senses of sight and hearing. The histologic picture of the neoplasm affecting the optic nerve head is described, and photomicrographs illustrating these changes are shown. Three years after the disease had been discovered in the left eye, signs of raised intracranial pressure developed due to a neoplasm or possibly to several neoplasms inside the skull. The right auditory nerve was involved. A subtentorial decompression was followed two days later by death.

W. ZENTMAYER.

RELATION OF THE SENSORY VISUAL CORTEX TO THE OCULOMOTOR CORTEX. E. CLAES, Compt. rend. Soc. de biol. **127**: 1116, 1938.

The experiments on cortical function were made on cats with the brain isolated by suboccipital section of the cord, a procedure which permits a truer physiologic response than general anesthesia. Each hemisphere has two oculomotor areas. The anterior, located in the sigmoid gyrus, produces when stimulated a bilateral and equal dilatation of the pupil, and the eyes move (up or down), reversing the position held before stimulation. The posterior area in the posterior portion of the suprasylvian gyrus on stimulation provokes a conjugate deviation to the opposite hemisphere. The posterior motor areas are mutually inhibitory, but the motor areas of the same side are synergistic, and the anterior motor areas are apparently unconnected with each other. Wake-

fulness and activity of the striate and auditory areas have each a tonic cerebral action and effect a lowering of the threshold stimulus of either oculomotor area.

J. E. LEBENSOHN.

Ocular Muscles

ON THE VALUE OF ORTHOPTIC TRAINING. F. W. LAW, Brit. J. Ophth. **22**: 193 (April) 1938.

There are special reasons why orthoptic training should be scrutinized as to results. The recent interest in this branch of ophthalmology has resulted in a large number of medically unqualified persons undergoing a course of study subsequent to undertaking the training of squinting children, which training is usually of a protracted nature. Aided by the somewhat apathetic acquiescence of the majority of ophthalmic surgeons, the practice of orthoptics from some points of view has reached the position of a vested interest, and its uncritical acceptance is all that is needed to insure its persistence, quite apart from its excellence.

Law gives a detailed analysis of 91 cases in which orthoptic training was used.

After a consideration of the facts obtained, the author arrives at the following conclusions:

A far greater proportion of persons with squint than is generally supposed can be cured by glasses and, when necessary, by occlusion.

Only those persons for whom such measures have failed should be submitted to orthoptic training.

Patients should be selected for training on the basis of clinical merits and with due consideration for the end result desired and for the justification for the means necessary to obtain it.

The results of orthoptic training are far less dramatic and far less certain than they are held to be in some quarters, and the descriptions of the value of such training which have appeared in the medical press have all erred on the side of optimism.

W. ZENTMAYER.

A STUDY OF MINER'S NYSTAGMUS. RAYMOND S. BROCK, Brit. M. J. **1**: 443 (Feb. 26) 1938.

This paper is based on a study of nystagmus in 15 pits of North Wales over a period of ten years. The nature of the ocular movements is studied, and the various theories as to their cause are discussed. The importance of psychologic factors is considered, and suggestions are offered, first, as to the prevention of the disability and, secondly, as to a more secure basis on which the disability may be assessed.

The author's conclusions are summarized as follows:

1. Prolonged work below ground may bring about oscillation of the eyes which does not necessarily cause disablement.

2. When disability does arise, it is usually due to an associated psychoneurosis.

3. The prevention of this depends on: (*a*) recognition of the foregoing facts; (*b*) the treatment of contributory disorders; (*c*) dimin-

ished stress in the mine; (*d*) the barring of unsuitable applicants; (*e*) improved economic conditions, and (*f*) stricter assessment of disablement.

ARNOLD KNAPP.

Pharmacology

EFFECT OF AN EXTRACT OF CILIARY BODY ON BLOOD PRESSURE. D. MICHAIL and P. VANCEA, Compt. rend. Soc. de biol. **127**: 453, 1938.

An extract of ciliary body was made according to the general technic advised by Roger for the preparation of organic extracts. The intravenous injection of a small amount produced a slight fall in blood pressure followed by a notable rise; injection of a larger amount caused a slowly progressive rise from the start. These changes were unmodified by previous injection of atropine or ergotamine but were increased by previous section of the vagus nerves.

J. E. LEBENSOHN.

EFFECT OF AN EXTRACT OF CILIARY BODY ON ISOLATED FROG HEART. D. MICHAIL and P. VANCEA, Compt. rend. Soc. de biol. **127**: 455, 1938.

The frog heart was isolated according to the technic of Straub and treated with an extract of ciliary body. Small doses produced a progressive depressive action, ending in complete ventricular arrest. Larger doses produced immediate cardiac arrest in diastole. In both instances reanimation ensued after irrigation with Ringer's solution.

J. E. LEBENSOHN.

Physiology

A STUDY OF DIET IN RELATION TO HEALTH: DARK ADAPTATION AS AN INDEX OF ADEQUATE VITAMIN A INTAKE. J. R. MUTCH and H. D. GRIFFITH, Brit. M. J. **2**: 565 (Sept. 18) 1937.

The association of night blindness with diet was accepted long before studies on vitamins were begun. With the rapid improvement in diet since the beginning of the century, obvious deficiency diseases have become more and more uncommon. As the important dietary sources of vitamin A are relatively expensive foodstuffs, it is evident that vitamin deficiency will be more common among the poorer people. Preliminary to a study of diet relative to health, tests have been made to discover the extent to which deficiency of vitamin A exists and the daily requirements of vitamin A to insure perfect health.

Given an accurate measure of the power of adaptation, the following two questions will arise: 1. To what extent does difference in the power of adaptation occur? 2. Can adaptation be improved by giving vitamin A?

The power of dark adaptation was estimated by Edmund's test type and Tscherning's photometric glass. A limit is shown below which all subjects react, with improved adaptation to administration of vitamin A. Those whose first performance is above this limit give no response to

the administration of vitamin A. The test is sufficiently simple to be applied to intelligent children of 6 years and so short that it is suitable for serial examination. The results are unaffected by practice.

ARNOLD KNAPP.

The Pupil

THE INCIDENCE OF UNEQUAL PUPILS IN UNCONVICTED PRISONERS. H. K. SNELL and G. A. CORMACK, Brit. M. J. **1**: 672 (March 26) 1938.

Two hundred and ninety-five of 3,000 unconvicted prisoners showed persistent anisocoria. No cause could be found in 139. An error of refraction was the only anomaly in 62. There was a history of syphilis in 30, and in the remaining 64 the inequality was due to various causes. The anomaly was associated with mental disease in only 4. The condition was therefore of no significance in approximately two thirds of the subjects, and in the others it was associated with syphilis and a history of injuries to the head, conditions which may be related to mental disorders. If laboratory tests are not available, anisocoria should be regarded with suspicion and may be of medicolegal significance.

ARNOLD KNAPP.

Refraction and Accommodation

TEMPORARY SPASMODIC MYOPIA DUE TO ARSPHENAMINE. E. DE C. FALCÃO, Ann. d'ocul. **174**: 847 (Dec.) 1937.

The introduction of arsphenamine into the human body is often followed by toxic conditions which manifest themselves in different forms. Of these complications, many have been observed and reports on them have been published; others have not been recorded, so that reports of new cases are always worthy of note.

The author reports what is to him an interesting case of arsphenamine intoxication. Spasmodic contraction of the ciliary muscle appeared in a patient 33 years of age after the intramuscular injection of arsphenamine. This caused a change in the refraction of the lens and transient acute myopia. A detailed description of the case is given.

The article closes with a summary of the previous case reports that have been published.

S. H. MCKEE.

INTRACAPSULAR ACCOMMODATION OF THE LENS. F. BOSA, Ann. di ottal. e clin. ocul. **65**: 621 (Aug.) 1937.

The author calculated the dioptric power of the lens according to a modification of Gullstrand's equation. He found it to be 20.28 diopters in the nonaccommodated lens. In the accommodated lens, without consideration of the increase in the total index of refraction, it was 28.30 diopters. When the increase in the total refractive index resulting from a change in the curvature of the nucleus was considered, the dioptric

power was increased to 32.25 diopters. The difference between the two latter figures, or 3.95 diopters, would then represent the increase in refractive power to intracapsular accommodation.

S. R. GIFFORD.

Retina and Optic Nerve

EDEMA OF THE RETINA. P. BAILLIART, Ann. d'ocul. **175:** 133 (Feb.) 1938.

From reading the ophthalmic classics one would think that edema of the retina is rare. It is characterized by an increase in the quantity as well as by an alteration in the makeup of the interstitial fluid. Edema naturally accompanies all hemorrhages, which are so frequently found in the center of the retina. There are many causes that bring on retinal edema. They may be circulatory or inflammatory and may originate within the tissues themselves. It must be understood that certain modifications, mechanical or chemical, in the composition of tissue make them capable of retaining an abnormal amount of interstitial fluid. According to Achard and Loeper and Widal and Lemierre, an excess of salt in the blood of an edematous person is deposited in the tissues. In some persons with swollen lids, edema of the retina is often seen without arterial hypertension. Achard believes that all chemical modifications of the interstitial fluid, whether they result in an abnormal amount of fluid or not, may provoke edema. Renal changes act in a similar manner. The action of cholesterol is also known.

The histologic constitution of the retina is less predisposed to edema than other tissues, as elsewhere there are zones of predilection. The author describes in detail some aspects of edema of the retina, the mechanism of the appearance of edema and the subjective symptoms.

S. H. MCKEE.

TREATMENT AND DEVELOPMENT OF TWO CASES OF THE SYNDROME OF GRÖNBLAD AND STRANDBERG (ANGIOID STREAKS IN THE RETINA AND PSEUDOXANTHOMA ELASTICUM). R. FRYDMAN, Ann. d'ocul. **175:** 154 (Feb.) 1938.

There are numerous articles on this subject, the most important of which is that of Grönblad and Strandberg. In a recent article, Franceschette and Roulet gave a general description of the disease and its relation to other conditions.

Two cases of this syndrome are described in detail, and a chart showing the results of the treatment by injections of calcium in the first case is presented.

Frydman concludes from the course of the condition in these 2 cases that calcium and vitamins B_1 and C as prescribed by Franceschette have given satisfactory results. This treatment has a remarkably rapid action, but its effect is nil in cases of lesions of long standing, and it does not prevent new hemorrhages from forming. Patients in whom angioid streaks of the retina occur have had repeated retinal hemorrhages. When a retinal hemorrhage has occurred, treatment by calcium and vitamin C should be begun. Such patients should receive a series of ten treatments every two months.

S. H. MCKEE.

THE ANGIOSCLEROTIC AND ANGIOSPASTIC RETINOSES. D. CATTANEO, Ann. di ottal. e clin. ocul. **65:** 721 (Oct.) 1937.

A number of forms of retinal disease are discussed which the author believes are caused by sclerosis or spasm of the retinal vessels. The literature on these conditions is reviewed, including that on circinate retinitis (Fuchs), senile macular exudative retinitis (Coppez and Danis), disciform degeneration of the macula (Kuhnt and Junius), central capillarospastic retinitis (Horniker) and retinal capillaritis (Bailliart). The close relation which exists between the first three conditions is emphasized, senile macular exudative retinitis and disciform degeneration of the macula being perhaps identical conditions which are often associated with the picture of circinate retinitis. The author believes these conditions to be based on organic changes in the arteries and the arterioles. The two last-named conditions described by Horniker and Bailliart represent the results of spasm affecting the small arterioles or capillaries of the macular region. At the onset there are edema and a paracentral scotoma. The edema disappears, and a number of white spots resembling Gunn's dots are left.

The author reports 17 cases in which the small lesions characteristic of capillaritis, or, as he prefers to call it, capillarosis, were combined with other changes due to involvement of the larger vessels. Complete studies were carried out in each case, including measurement of the retinal blood pressure with the dynamometer, determination of the capillary index according to the method of Fritz and in most cases endoscopic study of the circulation, the pressure which was required to obliterate the visible movement of corpuscles being recorded.

The small lesions characteristic of capillarosis were present in all cases. In 2, they were associated with central disciform degeneration; in 1, with senile cystic degeneration; in 1, with circinate retinitis, and in 2, with chronic glaucoma. The general systolic and diastolic blood pressure and the pressure in the retinal arteries were elevated in most but not in all cases. In 2 cases marked general and retinal hypertension were present, with no signs of arteriosclerosis, and in these cases the small lesions which were present must be considered due to vascular spasm. The lesions may best be interpreted as foci of degeneration due to an insufficient blood supply.

The author believes that the whole group of changes considered may well be named angiosclerotic and angiospastic retinoses and may be divided, according to location of the lesions, into three types. The macular type would include senile macular degeneration, senile exudative macular retinitis and central disciform degeneration. The paramacular type would include retinitis circinata. The disseminated type would include isolated and diffuse capillaritis.

A bibliography accompanies the article.

SANFORD R. GIFFORD.

PATHOGENESIS OF CHOKED DISK. F. SCHIECK, Arch. f. Ophth. **138:** 48 (Oct.) 1937.

Schieck performed the following experiment on a fresh clinically normal human eyeball with the whole orbital portion of the optic nerve attached to it. The specimen was obtained from a patient in whom a

recurrent sarcoma of the orbit necessitated exenteration. Immediately after the operation he injected india ink under moderate pressure into the intravaginal space of the optic nerve and then fixed, embedded and sectioned frontally the whole specimen. Microscopic examination of the sections revealed filling of the intravaginal space, or rather of the space situated between the inner surface of the dura and the pia covering the nerve. In addition, particles of ink extended along the outer surface of and into the strand of tissue which accompanies the central vessels, into the optic nerve. Except for this axial strand of tissue, the optic nerve was free of particles of ink. This experiment proves that "there is an open communication between the intravaginal space of the optic nerve and the tissue spaces of the axial strand." According to Schieck, in diseases which are associated with increased intracranial pressure the cerebrospinal fluid gains access to the distal portion of the optic nerve through this axial strand of tissue. No particles of ink were found in the orbital tissues outside the dura. Schieck, therefore, doubts the existence of the physiologic stream of tissue fluid which, according to Behr (*Arch. f. Ophth.* **134:** 249, 1937), goes from the intravaginal space by preformed channels into the orbital tissues.

P. C. KRONFELD.

Trachoma

TREATMENT OF TRACHOMATOUS PANNUS BY SUBCONJUNCTIVAL AUTOHEMOTHERAPY. DELORD, Bull. Soc. d'opht. de Paris, July 1937, p. 372.

The author reports the results obtained in the treatment of 6 patients with trachoma by means of subconjunctival autohemotherapy. From five to seven injections are advised at intervals of from ten to fifteen days, to allow for absorption.

The advantages of the method are listed as follows: It is simple, rapid, painless and efficient, and the patient need not be hospitalized. It is also analgesic and lessens photophobia.

L. L. MAYER.

TREATMENT OF TRACHOMA WITH TRACHOZID. L. KEPPICH-OLÁH, Klin. Monatsbl. f. Augenh. **99:** 234 (Aug.) 1937.

Trachozid, prepared at the Serotherapeutic Institute at Vienna, is a derivative made from animal toxin by a special process. Its action is nontoxic for the eye and the entire organism. The author corroborated the favorable results obtained by other writers after treating 100 patients with trachoma at the ophthalmic department of the Royal Hungarian Hospital at Gyula. All her patients, mostly with stubborn and protracted trachoma, were benefited. The pannus receded promptly; lesions and ulcerations of the cornea healed promptly, and the tendency of the pannus to recur was diminished. The duration of the treatment was considerably reduced, so that the patients could return to work sooner than after other therapeutic procedures. Hospitalization was not required.

K. L. STOLL.

Tumors

A Melanoma of the Iris with Pathological Findings. F. T. Tooke, Brit. J. Ophth. **22**: 153 (March) 1938.

In a man aged 24, a spot of pigment the size of a pinhead had appeared on the right iris seven years previously and had gradually increased in size until it extended from a point just beyond 9 o'clock at the pupillary margin below to within 2 mm. of the corneoscleral margin above, and upward from 11:30 o'clock below at the pupillary margin to the limitations of the root of the iris above. The mass was melanotic and slightly elevated. It was removed by iridectomy. A detailed description of the histologic features of the tumor is given, and a critical résumé of the opinions advanced to date regarding the derivation of the cells responsible for pigmented growths is given.

The author states that "these mesoblastic melanoblasts of the iris stroma have the appearance of mesoblastic cells as our bleached sections show and many on very good grounds would call them mesoblastic. It is true that they are 'dopa' positive (i. e., they contain a specific ferment for producing melanin from dihydroxyphenylalanine) which is an ectodermal characteristic. If it is true that they are mesodermal then we have another instance, of which in the eye there are many, of a phylogenetic aberration. These are cells which partake of the characteristics of both ectoderm and mesoderm. One might mention as another instance the development of the smooth muscle of the iris from ectoderm."

The article is illustrated.

W. Zentmayer.

Nevocarcinoma of the Lacrimal Caruncle: Clinical and Anatomic Study. F. Terrien and P. Veil, Arch. d'opht. **53**: 721 (Oct.) 1936.

The rarity of malignant tumors of the caruncle and the variety of their anatomic appearance make them difficult to diagnose clinically. The authors describe a case in which the patient was followed until death; autopsy was not performed. Photographs of the gross specimen and microscopic sections are presented. In spite of its relatively benign clinical appearance and immediate extensive excision, recurrence took place in two months. As the patient refused exenteration, at first radium and roentgen therapy were tried, but without benefit. Later operation was done. The inefficacy of radiotherapy in these cases is generally recognized. The authors affirm that exenteration done early is the best treatment. The discovery of the presence of nevus cells and malignant changes on histologic examination implies a particularly gloomy prognosis for life.

S. B. Marlow.

Treatment of Epithelioma of the Eyelids. Ducuing, Couadau and Lu-Van-Xuong, Arch. d'opht. **53**: 800 (Nov.) 1936.

This report, which is divided into five sections, deals with the therapeutic methods available for the treatment of epithelioma of the eyelids, their advantages and disadvantages, the opinions expressed in

the literature by other writers, the therapeutic indications according to their technic and the results obtained by them in 55 cases. In cases of circumscribed epithelioma of the skin, preference is given to radium by surface application alone or combined with the use of radium needles. In cases in which the epithelioma involves the free border of the lid, radiopuncture is advocated. When the commissures are affected, if the lesion is limited to the skin, radium needles alone or combined with plaques are used. If the conjunctiva or the lacrimal sac is affected, extensive excision followed by the use of radium is indicated. If the palpebral conjunctiva alone is involved wide excision of the involved lid is advocated. If the bulbar conjunctiva or the globe itself is affected, enucleation with emptying of the orbit is selected. If the bones are invaded, wide resection by means of electrosurgery avoids extensive hemorrhage. The results obtained with these various methods of treatment in the 55 cases are as follows: roentgen irradiation in 1 case, with cure; the diathermic needle in 1 case, with cure; radium therapy and surgical treatment in 3 cases, with cure in 2 and improvement in 1; surgical treatment alone in 4 cases, with cure; radium therapy in 46 cases with cure in 34 (73.9 per cent), improvement in 5 and failure in 5. Two of the patients in the last group were lost from observation. These results are compared with those reported by others. The percentage of cure varied from 66 to 97.

S. B. MARLOW.

GENERAL CONSIDERATIONS OF PSAMMOMAS APROPOS OF A CASE OF CYSTIC PSAMMOMA OF THE OPTIC PAPILLA. R. ARGAUD and A. COUADAU, Arch. d'opht. **53**: 869 (Dec.) 1936.

Psammomas of the optic nerve are rare, and cysts of the papilla are more rare. The authors report a case in which after the subsidence of an attack of acute glaucoma ophthalmoscopic examination revealed a brilliant yellowish mass containing small crystals covering the disk. A subsequent attack of glaucoma necessitated enucleation. The histologic structure of the mass is described in detail, and a diagnosis of psammoma was made. A brief review of the probable mode of formation of such tumors is presented.

S. B. MARLOW.

Therapeutics

FEVER THERAPY IN OPHTHALMOLOGY. L. HAMBRESIN, Ann. d'ocul. **174**: 721 (Nov.) 1937.

Although the attention of oculists has been called to the therapeutic use of induced fever, it is not employed as a rule in ophthalmic practice. Articles on this subject are still scarce, especially in the French language.

Hambresin discusses the essential difference between fever therapy and the therapeutics of shock, as the two methods are often confused, and the latter is particularly employed in the practice of ophthalmology. The majority of oculists use shock medication, the most popular form being the injection of whole milk. Pyretotherapy is the treatment of disease by raising the temperature of the body. The fever cures by rendering the ordinary therapeutic measures more active or by making

the organism more receptive to these. With good reason, C. Richet and A. Meyer-Heine stated that "shock is one thing, pyretotherapy is another." Therapeutic shock may produce good results, even without the elevation of temperature.

The writer concludes that pyretotherapy, or fever therapy, is useful in the treatment of simple atrophy of the optic nerve. The results are not permanent or perfect, but it seems to be the only treatment that halts the progress of the lesion. Certain specific pupillary conditions are also helped, and good results may be obtained in cases of syphilitic parenchymatous keratitis, keratitis, iritis and iridocyclitis of all kinds.

S. H. McKee.

Society Transactions

EDITED BY W. L. BENEDICT

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

JAMES W. WHITE, M.D., *Chairman*

March 21, 1938

RUDOLF AEBLI, M.D., *Secretary*

CHOROIDAL HEMORRHAGE SIMULATING A CHOROIDAL TUMOR. DR. MARTIN COHEN.

A. K., a woman aged 51, had a history of marked systemic hypertension for the past six or seven years, but otherwise she was essentially normal. The menopause occurred at the age of 49. At the time of the last examination the patient had considerable pallor of the skin and conjunctivas. The heart was slightly hypertrophied. The Wassermann reaction was negative, and the renal function was normal. The blood pressure was 290 systolic and 140 diastolic. The hemoglobin content was 70 per cent.

The patient was referred to me on Jan. 19, 1938, complaining of sudden excruciating pain in the left eye. Examination showed vision in the right eye to be 20/20. The fundus, aside from slight arteriovenous compression of the superior temporal vein above the disk, was essentially normal. Vision in the left eye equaled 20/20. With the exception of slight venous compression, the fundus appeared normal, but the ocular conjunctiva was markedly congested. Hot compresses, acetylsalicylic acid and absolute rest were prescribed with the object of alleviating the pain.

The patient returned the following day and reported that the severe pain still persisted. Slit lamp examination showed no iritic involvement, and the intraocular tension was normal. The previous treatment was continued, and a general physical examination was advised.

The pain in the left eye gradually subsided, and the patient returned two weeks later for reexamination, complaining of a cloud in front of the left eye. Vision in the right eye was now 20/30, and the fundus showed moderate edema of the temporal side of the disk. There were a few hemorrhages and lymphocytic infiltrations around the disk, with some dots of pigment in the periphery. Vision in the left eye had also diminished slightly (20/30). Ophthalmoscopic examination of this eye revealed a large, ovoid, flat blackish mass, about two and one-half times the size of the disk, in the fundus peripherally down and in, causing a scotoma above and temporally. The mass was slightly elevated, with an ill defined margin, and no vessels were visible on its surface. Transillumination was deficient over the dark area. The surrounding retina appeared hyperemic, and floating opacities could be seen in the vitreous. The intraocular tension was 18 mm. of mercury (Schiötz) in each eye.

Recent examination of the right eye showed further diminution of vision (20/70). There were engorgement of the retinal veins and slight contraction of the arteries, with prominence of the central light reflexes; the smaller arteries were not perceptible. The outline of the disk was evident on all but the temporal margin, which was edematous. Several fresh hemorrhages bordered the venous branches; the lymphocytic infiltrations were more numerous, and the dots of pigment which resulted from previous hemorrhages were being absorbed. Vision in the left eye, which had at one time been reduced to 20/50 had improved to 20/30. The opacities of the vitreous had been largely absorbed, although the optic disk could not yet be perceived. The large blackish mass was now steel gray, owing to a commencing organization of the blood clot. Transillumination showed that the mass was contracting, and its outline at the upper margin was becoming evident. The surrounding retina appeared hyperemic; the retinal vessels were distinctly visible.

The diagnosis on the basis of the fundus picture was arteriolosclerosis of the right eye, and localized choroidal hemorrhage in the left eye. The general diagnosis was arteriolosclerosis and essential hypertension of a severe type.

The treatment consisted of absolute rest and general measures for the systemic hypertension.

Conclusion.—The excruciating pain in the left eye was probably caused by the localized pressure of the encapsulated blood clot on the surrounding neural elements. Choroidal hemorrhage has seldom been observed and reported in cases of disease of the vascular walls. It is difficult, therefore, to state definitely whether there is an underlying growth. However, from the progress of the condition, the accompanying systemic condition and the degeneration of the vascular walls in the opposite eye, it is inferred that the lesion in the left eye is probably a choroidal hemorrhage due to a venous thrombosis rather than a choroidal tumor.

DISCUSSION

DR. LEWIS WEBB CRIGLER: Several years ago I reported before this section a case of subchoroidal hemorrhage in which a diagnosis of sarcoma of the choroid had been made by three ophthalmologists. Not until the enucleated eye was sectioned and examined microscopically was the true diagnosis made. Fortunately for both the patient and the oculist, the eye had to be removed on account of extreme pain and tension. All preception of light had been lost.

Investigation revealed that the patient was a free bleeder. At one time after a slight fall there had been a profuse subcutaneous hemorrhage between the knee and the hip. At another time after the extraction of a tooth there was bleeding from the socket for several days. With this experience before me, I have been constantly on guard to avoid a similar mistake.

Several months ago a patient 70 years of age was referred to me by Dr. Beck, of New Rochelle. Dr. Beck had seen this patient just a week previously and found both fundi normal except for mild signs of vascular sclerosis. On her second visit he found the vision in the left eye quite blurred, and not being satisfied with the result of his examination he

referred her to me for consultation. There was no external evidence of inflammation. The anterior segment of the eye was normal, and tension was 18 mm. of mercury. The pupil was dilated for examination of the fundus. The lens was clear and the vitreous turbid. By means of oblique focal illumination and a B B loupe a distinct elevation of the choroid and retina was seen in the inferonasal quadrant far forward near the ora serrata. The posterior aspect of the detachment could not be seen on account of the turbid vitreous. Transillumination over the detachment was dark.

The patient has been under observation by Dr. Beck and myself at weekly intervals. The tension has remained subnormal, and the vitreous is becoming less cloudy, but the detachment shows no material change. We hope to be able to report further details of this case at a later date. Whether the detachment is due to a transudate or to hemorrhage is a problem yet to be solved. In view of the clearcut history of the case, we think that a diagnosis of sarcoma can be excluded.

Dr. Cohen has had me see his patient with him on several occasions. The similarity of the picture presented in these 2 cases and the rarity of nontraumatic detachments of the choroid justify their presentation at this time.

Dr. August Leo Beck, New Rochelle: The patient mentioned by Dr. Crigler is an unmarried woman 70 years of age. She was first seen in 1916, when she was 48 years old, at which time she was given a presbyopic correction. The fundi and the corrected vision were normal. She was again examined in 1919, and the glasses were changed. The fundi and the corrected vision were normal. She was examined in 1921, the results of the examination being the same as those of the preceding one. The glasses were changed. She was next examined in 1925, at which time the glasses were changed. She had a mild conjunctivitis, but otherwise the findings were the same as at the previous examination. She was examined in 1936, when she was 67 years old. The glasses were again changed. Her corrected vision was normal, 20/20. There was a moderate sclerosis of both crystalline lenses, with barely preceptible visible opacification in the nucleus of the left lens. Examination of the fundi showed moderate angiopathic sclerosis in the retinas, with minute degenerative specks between the disk and the macular region of the left fundus.

The patient was next seen on Dec. 27, 1937, complaining of thread-like or filamentous specks before the left eye of several weeks' duration. There was a leash of injected conjunctival vessels on the nasal side of the left eye, which is still present. The right fundus was normal, except for the moderate angiopathic sclerosis. The left fundus was hazy, because of cloudiness of the vitreous. The vessels were visible; no hemorrhages were seen. No other abnormalities were noted, and the tension was not elevated. Potassium iodide was prescribed.

The patient was again seen on January 4, eight days after the last examination. During this interval the vision of the left eye was much more impaired. All fields were blocked out, except the lower one. No fundus reflex was present except from the extreme upper nasal field. The tension was low. A tentative diagnosis of retinal detachment was made. The patient was seen at this time in consultation with Dr. Crigler, who regarded the condition as choroidal detachment due to hemorrhage.

The Wassermann reaction was negative.

The condition resembles nothing so much as an intraocular new growth, and such a diagnosis would have been made but for the foregoing observations. These, with the unvarying softness of the eye and the rapidity of involvement, make it reasonably certain that we are not dealing with a new growth.

The underlying general pathologic process in this case is not cardionephritis. According to the patient's family physician, there has never been any nephritis. There is, however, definite cardiovascular disease of some years' duration. The blood pressure has often been 180 or well over. The heart is enlarged, and on numerous occasions there has been a variation or difference in the blood pressure readings on the two arms. For example, the pressure has been read as normal on the left arm and as from 170 to 180 on the right arm on the same occasion. The patient is highstrung and overconscientious in temperament. The Wassermann reactions are negative. Chemical examination of the blood has not been done, chiefly for economic reasons.

RETINAL DETACHMENT WITH CYST FORMATION: REPORT OF A CASE. DR. WALTER P. GRIFFEY (by invitation).

This case of retinal detachment is presented because of the interesting history given and the fact that the visual acuity has remained normal in the presence of such widespread pathologic change and the associated cystic formation.

W. B., aged 31, a deckhand on board a tugboat, had no complaints referable to his eyes. During the routine physical examination he was told that the vision in the right eye was defective and was advised to obtain glasses. In the course of refraction, retinal detachment with a cystic formation was observed in the left eye, and the patient was admitted to the hospital.

The patient had enjoyed excellent health in the past; his habits had been regular. Alcohol and tobacco had been used moderately. He stated that this was the first time that he had ever been under the care of a physician. Two months before admission he recalled having been struck across the nose and the left side of the face by rope line. He continued at work. There was no change in the function of the left eye, nor were any external signs of trauma observed.

General physical examination revealed a robust man of medium stature with no abnormalities other than the condition of the eyes. Examination showed the vision in the right eye to be 20/50, which was improved to 20/20 with correction of astigmatism. The fundus was normal. Vision in the left eye was 20/20. Externally the eye was normal. On ophthalmoscopic examination floating opacities could be seen in the vitreous. A retinal detachment sharply demarcated by a zone of choroiditis extended from the disk temporally immediately below the macula to end at the periphery near 3 o'clock. On the nasal side the detachment was not so acutely limited but faded away in the upper quadrant from 10 to 12 o'clock. A translucent cyst, spherical in outline and about 3 disks in diameter, was noted near the ora serrata between 7 and 8 o'clock. A second cyst, still farther forward and flat

in appearance, about 6 disks long and about 2 disks wide, was seen extending in an equatorial direction between 9 and 11 o'clock.

The visual field showed an almost complete defect in the upper half.

Urinalysis gave negative results. The Wassermann and Kahn reactions of the blood were negative. Repeated fecal analyses were negative for ova. The blood was found to be normal on study, and the differential counts were normal.

The problem of the etiologic basis, the treatment and the prognosis of this condition arises. Is the cyst the sole cause of the detachment? Could the trauma alleged have played a part in the cystic formation and detachment, and what outcome could be predicted on aspiration of the cyst and successful readjustment of the retina by operative means?

REPAIR OF AN OLD LACERATION AT MEDIAL END OF THE LOWER LID. DR. WENDELL L. HUGHES.

(The plastic repair reported here was illustrated by a motion picture film).

This method of plastic repair is presented because of the frequency of the occurrence of this type of laceration across the canaliculus at the medial end of the lower lid. The method of replacement gives uniformly good results. The problems presented are the same whether the laceration is recent or old or is healed in an improper position. In a laceration of this type the torn section of lid usually retracts to give the appearance of an actual loss of tissue. However, if the end is picked up and stretched it will usually bridge the defect. In an improperly healed old laceration the incision is reopened. A tongue of tissue is prepared at the nasal end of the tarsus and pulled upward, nasally and as far backward as possible by a double-armed suture on large curved needles, which are entered nasally behind the normal attachment of the torn medial end of the canthal ligament and emerge above the canthus on the side of the nose. The sutures are tied over a small piece of gauze externally. The conjunctiva and edges of the skin are approximated with fine sutures. A pressure dressing is applied and left in place for one week.

A PRELIMINARY REPORT OF THE VALUE OF FUNCTIONAL VISUAL TESTS FOR THE LOCALIZATION OF SUPRATENTORIAL TUMORS OF THE BRAIN. DR. CHARLES A. ELSBERG and DR. H. SPOTNITZ (by invitation).

In order to learn whether functional visual tests are of value in the localization of tumors and other diseases of the brain, tests were made to determine the visual thresholds, the relations between area and light intensity required for threshold vision and the significance of dark adaptation and the refractory period in healthy persons and in those suffering from intracranial lesions.

The tests were made in a dark room with simple apparatus. The objects used for the visual tests consisted of white squares of different sizes on a black background and of white squares with inner black squares as objects with internal contrast. The test objects were illuminated by red light and were viewed from a distance of 3.75 meters through a red Wratten filter.

When white squares from 9 to 40 mm. in size were used, the product of the cube root of the area and the threshold light intensity was found to be a constant. When squares of 40 mm. with internal contrast were used, the square root of the area multiplied by the light intensity was a constant, and when objects of 9 mm. were used, the area multiplied by the light intensity was also a constant. For all objects with internal contrast, the square root of the black area divided by the light intensity was a constant.

These varying relations between area and the minimum of light required for vision were found to be due to the quantitative functional relation between the cones, bipolar cells and ganglion cells of the retina. A formula was used which expressed the quantitative relations between area and light intensity for all of the test objects that were used and which showed the relative significance of excitation and of inhibition in vision.

The tests made it possible to study the changes that occur in dark adaptation and to show that the leveling of visual sensibility which occurs in passing from light to darkness is the result of the relative refractory period and the reciprocal relations between the intensity of the visual stimulus and the receptors in the retina and in the visual areas of the brain. It was found that when one eye is exposed in succession to a bright and a dim light, the duration of the refractory period is always longer than when the two eyes have been exposed in succession to a bright and a dim light and the test object is viewed with both eyes. In monocular vision after binocular stimulation by bright light the duration of the refractory period is regularly longer than in binocular vision after binocular stimulation and shorter than in monocular vision after monocular stimulation. The results of these tests showed that in so-called dark adaptation the central factor is of great importance.

By means of the tests it was found to be possible to determine the relative thresholds of vision in different parts of the fovea in healthy persons. Furthermore, in persons with diseases of the retina, and more particularly in those with papilledema and with primary atrophy of the optic nerve due to tumor of the brain, we were able to ascertain whether the central or the more peripheral parts of the fovea were more affected.

After a large number of healthy persons had been tested, patients with tumors and other lesions of the brain were studied in a similar manner. Up to the present time about 180 patients have been examined.

The functions of the two eyes were compared by means of tests of the monocular vision of each eye. The experiences thus far gained appear to indicate that in cases of tumor which cause pressure on the extracerebral visual pathways (optic nerves, chiasm and optic tracts) the threshold is raised, but the duration of the relative refractory period may be normal. When the growth is within the substance of the brain, the duration of the refractory period produced by successive exposure to a bright continuous light and to a dim light is prolonged in the contralateral eye. Therefore, it was possible by means of these functional visual tests to determine the hemisphere which was diseased. Furthermore, the results of stimulation by flickering light appeared to indicate that the frontal lobes of the brain may inhibit the visual functions of the occipital and parietal lobes. The presence or absence of this

inhibition often made it possible to conclude whether or not the lesion was in the frontal lobe of one or the other side.

Up to the present time our experiences appear to indicate that functional visual tests are important in the localization of diseases of the brain, but a much larger experience must be gained before the practical clinical importance of the tests can be evaluated.

DISCUSSION

DR. JOHN M. WHEELER: Dr. Elsberg has a wealth of experience in the diagnosis of intracranial lesions and in surgical work on the brain and in performing autopsies. After the eye has been exposed to a bright light and the person whose eye has been so exposed attempts to look at a dimly illuminated test object across the room, there is a delay in the period of recognition of that test object because of the previous exposure of the eye to light. This delay the authors have called the dark adaptation time, referring to the bright illumination of the exposure of the eye as light adaptation, and they have used synonymously the terms "duration of fatigue" and "duration of recovery" and "time of adjustment for the subject to see the test object" across the room, after he has been influenced by the exposure of the eye to bright light. They have capitalized on the dark adaptation phenomenon as an aid in the diagnosis of intracranial disease and particularly in the localization of intracranial tumors above the tentorium. In reference to lesions of the frontal lobe, the point they have capitalized on is that a pathway exists (almost definitely established) that leads from the frontal lobe of one side to the occipital lobe of the other side. The existence of this pathway has been used as an aid in the localization of these lesions.

The authors have established this important fact: The period of recovery from the exposure to the bright light (the dark adaptation time) is governed largely by the brain. The eye has something to do with it, but, according to their findings, the brain has more to do with it, and there are several evidences of it. Perhaps one of the best is that if one eye is exposed to bright light, the dark adaptation period is delayed in the other eye. As there is no direct communication between the two eyes anterior to the external geniculate ganglions, the authors figure that the dark adaptation phenomenon is governed more centrally than ocularly. That is rather disturbing to men who have spent large parts of their lives trying to determine the factors that influence a change from visual purple to visual red in the retina, but I think that it is well for one to think of the eye as an important part of the anterior portion of the brain and in this connection to think of the intracranial part as the governing part. Dr. Elsberg and Dr. Spotnitz, in the December 1937 issue of the *Bulletin of the Neurological Institute of New York*, refer to Dr. Ames' work. They stated: "Ames has shown interest in errors of refraction that may result in incomplete fusion of the cortical images, and this may be the cause of a variety of disturbances." I should like to point out that incidentally Ames has been interested in refraction, because correction of refraction is a help in the relief of symptoms, but that has not been his main interest. He has been interested in the difference in the size of the images in the two eyes, and he does not know—he says he does not know—where that difference is created, but he thinks that it is in the eye. I should like to suggest that it never has

been proved that the difference originates in the eye. It may arise in the brain, and it may be that the method of Ames could be used in the localization of tumors of the brain. The work of Dr. Elsberg and Dr. Spotnitz has been on foveal vision and dark adaptation in relation to foveal vision. I dare say some hyperscientific men might criticize their methods. They have gone ahead and have not been daunted by obstacles and have accomplished something in a relatively short time. Dr. Elsberg has talked to me about this work on several occasions, so I have tried to follow it.

I should like to make a suggestion: The effect of bright light on other parts of the retina might be important, and it may be that light adaptation (the exposure of the eyes to bright light) may have an important influence in bringing out defects in the visual fields. The aid of the ophthalmologist is of importance and should be of much greater importance in the diagnosing of intracranial lesions and their localization. The use of the ophthalmoscope has been important, the plottings of diplopia have been important and especially important are the plotting of the visual fields, so important that in some cases localization of a tumor of the brain has been dependent almost entirely on the defects in the visual fields. It may be that exposure to light as a preliminary to taking the visual fields may be helpful. That is just a suggestion, and I should like Dr. Elsberg to tell, if he will, what part the ophthalmologist is liable to play in carrying out of the ideas that he and Dr. Spotnitz are establishing in relation to the diagnosis of tumors of the brain, and whether he feels, as I do, that the effect of exposure to bright light may have an important influence on the visual fields, so that one may be led to diagnose earlier defects in the visual fields.

DR. CHARLES A. ELSBERG: What Dr. Wheeler has said regarding Dr. Ames' investigation is, I think, of great importance, because, as Dr. Wheeler mentioned, Ames' work had reference to the difference in size of the images. In binocular vision, images must be fused. The only place where they can be fused is centrally, and therefore the fact of Ames' investigation is another indication that the essential process in fusion and dark adaptation must be central. It is tremendously interesting that Dr. Wheeler should have picked up something that Dr. Spotnitz found in testing persons (with tumor of the temporal lobe, I think) who had no gross defects of the visual fields, that is, no defects that could be determined by perimetric examination. As the patients recovered after exposure to the bright light, they saw the test object first on the good side and not on what should have been the side with the hemianopic defect. Exactly what Dr. Wheeler predicted might happen has happened in a few isolated instances. It may be of value in cases in which there is a possibility of a hemianopic defect in the visual field, first to expose the subject to a brighter light and then to ascertain the visual fields. I think that it is probable that this procedure which Dr. Wheeler has suggested, and which has actually been tried in a few instances, will be of advantage to the ophthalmologist in the localization of lesions of the brain in examinations of vision. The method of examination and the apparatus to be used are both simple.

Book Reviews

Eleventh Annual Report of the Memorial Ophthalmic Laboratory. Giza, Cairo, Egypt: Schindler's Press, 1937.

The eleventh annual report of the Memorial Ophthalmic Laboratory presents a record of the administration and an account of the scientific work carried out during 1936. The activities of the laboratory are considered as follows:

1. Postgraduate education. Since the laboratory was opened ten years ago, one of its functions has been to provide a postgraduate course of instruction in ophthalmology for all doctors entering the ophthalmic service of the Ministry of Public Health. The teaching is essentially practical, and special attention is devoted to those features of ophthalmic practice peculiar to Egypt.

2. Pathologic section. Five hundred and fifty-four pathologic specimens were submitted for examination. Of the 125 globes included, 84, or 64 per cent, had been destroyed through acute ophthalmias. This is 13 per cent less than the figure for ten years ago, and the reduction is believed to be a result of the activity of the government ophthalmic hospitals.

A total of 22 specimens are described in detail and illustrated with photomicrographs. Of outstanding interest is a case of bilharzial granuloma, the third seen at the laboratory. In each instance the patient was a young child with clinical symptoms strongly resembling infection of the conjunctiva with Streptothrix. Other interesting specimens include one of early sarcoma of the bulbar conjunctiva, one of cartilage in the conjunctiva, unassociated with dermoid tumor, and one of "hyaline" bodies in the caruncle.

3. Clinical section. The clinical material studied at the laboratory appears to have been unusually varied. Five cases of special interest are reported in full, and the colored drawings which accompany 4 of them clearly illustrate the special features which they represent. These include a case of pemphigoid infection of the conjunctiva, a case of spring catarrh associated with epithelial dystrophy of the cornea, a case of xanthomatosis bulbi associated with cerebral signs, a case in which interesting macular changes occurred in association with cilioretinal anastomosis and a case of tumor of the pituitary gland with typical changes in the visual fields and unusual neurologic signs. A number of other interesting cases, including 1 of fly-blown orbit and 1 of infection of the conjunctiva with Streptothrix, are illustrated by photographs.

4. Research section. As in former years, the cause of trachoma and the epidemicity of the acute ophthalmias have been problems for special attention. This year's work also included a study on spring catarrh.

The studies on trachoma include a "Note on Free Initial Bodies and Free Elementary Granules in Trachoma of Egypt, Madras and Hong Kong" by F. H. Stewart, the pathologist of the laboratory. Up to the end of 1935 free initial bodies (Lindner) have not been found by

Stewart in Egypt, nor were free elementary bodies found in sufficient number to allow of certain identification. During 1936 he studied 37 cases of early trachoma in infants all under 1 year of age; free initial bodies were found in 7 and free elementary bodies in 3.

A second study on the inclusion bodies of trachoma is reported by R. P. Wilson, director of the laboratory. At Bahtim Village, near Cairo, he was able to examine 39 infants for inclusion bodies at weekly intervals from birth. From his results he concludes that inclusion bodies are present in 100 per cent of the patients at the onset of the disease and that they may be found before trachoma can be diagnosed clinically.

Wilson's observations on the acute ophthalmias have been continued, both at Giza and in other parts of Egypt. His findings indicate that conjunctivitis due to the Koch-Weeks bacillus assumes epidemic proportions when the mean maximum temperature reaches approximately 25 or 26 C. (77 or 78.8 F.), while gonococcic conjunctivitis becomes epidemic only at temperatures from 32 to 35 C. (89.6 to 95 F.). The two principal fly-breeding seasons in Egypt are spring and autumn; during the hottest months of the summer the incidence of flies is reduced. Koch-Weeks conjunctivitis becomes epidemic soon after the flies begin to increase and reaches a maximum coincidentally with the height of the spring fly-breeding season; gonorrheal conjunctivitis, on the other hand, does not increase in incidence during the first fly-breeding season until the temperature reaches approximately 32 C. The summer minimum of Koch-Weeks conjunctivitis and the August decline in gonorrheal conjunctivitis coincide with the summer minimum incidence of flies. The maximum incidence of gonorrheal conjunctivitis and the height of the autumnal rise in Koch-Weeks conjunctivitis coincide with the second fly-breeding season. Wilson has therefore concluded that although factors of direct contagion by hand, clothing, etc., cannot be neglected, heat and flies are the two most vital factors in the epidemicity of conjunctivitis in Egypt.

An extensive report by F. M. Lyons entitled "Observations on the Pathogenesis of Spring Catarrh" is of distinct interest. Lyons calls particular attention to the characteristic sticky palpebral membrane in the disease, for its diagnostic importance and for the light it throws on the nature of the disease. In cases of mild involvement the membrane is not always apparent on everting the lid but may be readily produced by various forms of irritation. Anything which gives rise to congestion and dilatation of the capillaries favors this exudation, which is accompanied by intense itching. Conversely, the formation of the membrane may be inhibited by any procedure which causes constriction of the capillaries, such as the application of ice or strong solutions of epinephrine hydrochloride. Freshly teased preparations and paraffin sections of the membrane were examined microscopically and found to consist of fibrin, mucin, some epithelial cells and considerable numbers of eosinophils. Lyons therefore concludes that the membrane is composed of two parts: (*a*) the true exudation of fibrin and blood cells, which must necessarily come from the subconjunctival capillaries, and (*b*) the mucin, a hypersecretion of the conjunctival goblet cells. He considers that the usual conception of the pathologic picture of spring catarrh is erroneous and that the lesions are due, not, as formerly

thought, to proliferation of preexisting connective tissue but rather to the invasion of the conjunctiva by new fibrinous material and wandering cells. He concludes that the whole picture of spring catarrh results from an abnormal exudation of fibrin and blood cells from the capillaries of the conjunctiva, and his results indicate that a faulty calcium metabolism is not a primary cause of this capillary dysfunction.

The eleventh annual report meets the standard set by previous numbers and constitutes a most valuable addition to the ophthalmic literature.

PHILLIPS THYGESON.

Citric Acid Studies Referring to the Eye. By Herman Grönvall. Paper. Pp. 279, with 50 tables. Copenhagen: Levin & Munksgaard, 1937.

This monograph, published as the fourteenth supplement of the *Acta Ophthalmologia,* is concerned chiefly with the occurrence of citric acid in the intraocular fluids. It begins with a discussion of the application and limitations of the Thunberg enzymatic method for the determination of citric acid. The different qualitative tests which confirm the presence of the acid in ocular fluids and tissues are reported. The quantity of citrate in the intraocular fluids and serums of various mammals, birds and fish, of bovine fetuses and also of patients suffering from different ocular diseases is recorded. It also deals with the relation of blood, tears, corneal permeability and retinal dehydrogenases to the metabolism of citric acid.

Citric acid is found in intraocular fluid in all mammals, birds and fish, although the amount in birds is higher than in other animals. Unlike dextrose, citric acid is evenly distributed throughout the vitreous humor. In fetal life the citrate content of the intraocular humors slowly declines until birth. After intravenous injection of citrate, the amount in the aqueous humor rises and falls with that of the blood serum. The citrate in the serum seems to be linked chemically or physically with protein. In cases of iridocyclitis, glaucoma and melanosarcoma of the choroid and after paracentesis of the anterior chamber the aqueous humor contains an abnormally large quantity of citric acid. There is no quantitative difference between the aqueous humor of cataractous and that of aphakic eyes. If the eye is kept in the dark, the amount of acid in the aqueous and vitreous humor diminishes.

The cornea shows an irreciprocal permeability to citrate which is dependent on the integrity of the epithelium. The corneal endothelium retards the diffusion of the salt less than does the epithelium.

Citric acid seems to be an intermediate product in metabolism of the ocular tissues. In the retina a dehydrogenase plays a part in this metabolism.

This well written monograph is the result of a carefully planned study of a special phase of ocular metabolism. More work of this kind is needed in ophthalmology.

ARLINGTON C. KRAUSE.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the Secretariate, 66 Boulevard Saint-Michel, Paris, 6e.

International Ophthalmologic Congress

Secretary: Dr. E. Marx, Costzeedijk 316, Rotterdam, Netherlands.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 33 Welbeck St., London, W., England.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. Stewart Duke-Elder, 59 Harley St., London, W. 1.
Secretary: Dr. Thomasina Belt, 13 Mitchell Ave., Jesmond, Newcastle-on-Tyne.
Place: Plymouth. Time: July 20-22, 1938.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. W. Lohlein, Berlin.
Secretary: Prof. E. Engelking, Heidelberg.
Place: Heidelberg. Time: July 4-6, 1938.

Midland Ophthalmological Society

President: Dr. W. Niccol, 4 College Green, Gloucester, England.
Secretary: T. Harrison Butler, 81 Edmund St., Birmingham, England.
Place: Birmingham and Midland Eye Hospital.

Ophthalmological Society of Egypt

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
All correspondence should be addressed to the Secretary, Dr. Mohammed Khalil.

Ophthalmological Society of the United Kingdom

President: Mr. Gordon M. Holmes, 9 Wimpole St., London, W. 1.
Secretary: Mr. H. B. Stallard, 35 Harley St., London, W. 1.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Time: July 7-9, 1938.

PALESTINE OPHTHALMOLOGICAL SOCIETY

President: Dr. Arieh Feigenbaum, Abyssinian Str. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel-Aviv.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. Malcolm Hepburn, 111 Harley St., London, W. 1, England.
Secretary: Dr. C. Dee Shapland, 15 Devonshire Pl., London, W. 1, England.

SOCIÉTÉ FRANÇAISE D'OPHTALMOLOGIE

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7è.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. K. G. Ploman, Stockholm, Sweden.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

TEL-AVIV OPHTHALMOLOGICAL SOCIETY

President: Dr. D. Arieh-Friedman, 96 Allenby Str., Tel-Aviv.
Secretary: Dr. Sadger Max, 9 Bialik Str., Tel-Aviv.

TSINAN OPHTHALMOLOGICAL SOCIETY

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.
Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. S. Judd Beach, 704 Congress St., Portland, Maine.
Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.
Place: St. Louis. Time: May 15-19, 1939.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Washington, D. C. Time: Oct. 9-14, 1938.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Frederick Tooke, 1482 Mountain St., Montreal, Canada.
Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Place: Hot Springs, Va.

CANADIAN OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Gordon M. Byers, 1458 Mountain St., Montreal.
Secretary-Treasurer: Dr. Alexander E. MacDonald, 421 Medical Arts Bldg., Toronto.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Secretary: Miss Regina E. Schneider, 50 W. 50th St., New York.

SECTIONAL

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. C. Gardner, 11 N. Main St., Fond du Lac.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. Edwin B. Goodall, 101 Bay State Rd., Boston.
Secretary-Treasurer: Dr. Trygve Gundersen, 243 Charles St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. F. C. Cordes, 384 Post St., San Francisco.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.
Place: San Francisco. Time: June 19-22, 1939.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. L. Goss, Cobb Bldg., Seattle, Wash.
Secretary-Treasurer: Dr. Purman Dorman, 1115 Terry Ave., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. L. J. Friend, 425 E. Grand Ave., Beloit, Wis.
Secretary-Treasurer: Dr. Thorsten E. Blomberg, 501–7th St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Don M. Howell, Alma, Mich.
Secretary-Treasurer: Dr. Louis D. Gomon, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. R. A. Kelly, 304 N. Main St., Mitchell, S. D.
Secretary-Treasurer: Dr. J. C. Decker, 515 Frances Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. John Hunter McRae, 26 Sheldon Ave., S. E., Grand Rapids.
Secretary-Treasurer: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. M. Harris, Johnstown.
Secretary-Treasurer: Dr. C. Wearne Beals, 41 N. Brady St., DuBois.

STATE

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. John C. Long, 324 Metropolitan Bldg., Denver.
Place: Capitol Life Bldg., Denver. Time: 7:30 p. m., third Saturday of each month, October to May, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. Charles T. Flynn, 41 Trumbull St., New Haven.
Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. Grady E. Clay, 384 Peachtree St., N. E., Atlanta.
Secretary-Treasurer: Dr. J. Mason Baird, 511 Medical Arts Bldg., Atlanta.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. C. W. Rutherford, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Marlow W. Manion, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: First Wednesday in April.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. H. Lamb, American Bank Bldg., Davenport.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.
Place: Davenport.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. Francis E. Le Jeune, 632 Maison Blanche Bldg., New Orleans.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Place: Gulfport, Miss. Time: May 8, 1939.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. F. N. Smith, Grand Rapids Clinic, Grand Rapids.
Secretary: Dr. Dewey R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

Minnesota Academy of Ophthalmology and Otolaryngology

President: Dr. Frank N. Knapp, 318 W. Superior St., Duluth.
Secretary-Treasurer: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Roy Grigg, Bozeman.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. Norman W. Burritt, 30 Beechwood Rd., Summit.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: Atlantic City. Time: June 1939.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Algeron B. Reese, 73 E. 71st St., New York.
Secretary: Dr. Chester C. Cott, 333 Linwood Ave., Buffalo.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. M. Lilly, 302 Old St., Fayetteville.
Secretary-Treasurer: Dr. Frank C. Smith, 106 W. 7th St., Charlotte.
Place: Charlotte. Time: October.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. N. A. Youngs, 322 De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, 516-6th St., Valley City.
Place: Fargo. Time: May 1939.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. S. B. Fishburne, 1430 Marion St., Columbia.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.
Place: Columbia. Time: Nov. 1, 1938.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Kate Savage Zerfoss, 165–8th Ave., N., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 805 Medical Arts Bldg., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Charles T. St. Clair, 418 Bland St., Bluefield, W. Va.
Secretary-Treasurer: Dr. M. H. Williams, 30½ Franklin Rd., S. W., Roanoke.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Andrew Rados, 31 Lincoln Park, Newark.
Secretary: Dr. William F. McKim, 317 Roseville Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Akron Academy of Ophthalmology and Otolaryngology

President: Dr. L. E. Brown, Second National Bldg., Akron.
Secretary-Treasurer: Dr. C. R. Anderson, 106 S. Main St., Akron.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. J. Mason Baird, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. Alton V. Hallum, 478 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Frank B. Walsh, Wilmer Institute, Johns Hopkins Hospital, Baltimore.
Secretary: Dr. Fred M. Reese, 6 E. Eager St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. E. Clifford Place, 59 Livingston St., Brooklyn.
Secretary-Treasurer: Dr. Frank Mallon, 1135 Park Pl., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Ivan J. Koenig, 40 North St., Buffalo.
Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Georgiana Dvorak-Theobald, 715 Lake St., Oak Park.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Oto-Laryngological Club

President: Dr. Clarence Engler, 2323 Prospect Ave., Cleveland.
Secretary: Dr. Fred Dixon, 2060 E. 9th St., Cleveland.

Cleveland Ophthalmological Club

Chairman: Dr. Paul Moore, Republic Bldg., Cleveland.
Secretary: Dr. G. Leslie Miller, 14805 Detroit Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Clerk: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Hugh G. Beatty, 150 E. Broad St., Columbus, Ohio.
Secretary-Treasurer: Dr. W. A. Stoutenborough, 21 E. State St., Columbus, Ohio.
Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. Edgar G. Mathis, 416 Chaparral St., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 416 Chaparral St., Corpus Christi, Texas.
Time: Second Thursday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Lester H. Quinn, 4105 Live Oak, Dallas, Texas.
Secretary: Dr. J. Dudley Singleton, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. William Fowler, 1066 Maccabee Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. L. A. Hulsebosch, 191 Glen St., Glen Falls.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. R. A. Gough, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. Charles R. Lees, 806 Medical Arts Bldg., Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 116 E. Fulton St., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Louis Daily, 1215 Walker Ave., Houston, Texas.
Secretary: Dr. Herbert H. Harris, 1004 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. J. K. Leasure, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to June.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. E. N. Robertson, Concordia, Kan.
Secretary: Dr. John S. Knight, 1103 Grand Ave., Kansas City, Mo.
Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Paul Nilsson, 211 Cherry Ave., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Clifford B. Walker, 427 W. 5th St., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

LOUISVILLE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Gaylord C. Hall, Brown Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Charles K. Beck, Starks Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Earle Breeding, 1801 I St., N. W., Washington.
Secretary: Dr. Elmer Shepherd, 1606–20th St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sonders, Medical Arts Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. John E. Mulsow, 231 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. John B. Hitz, 411 E. Mason St., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

MINNEAPOLIS OPHTHALMOLOGICAL SOCIETY

Chairman: Each member, in alphabetical order.
Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

MONTGOMERY COUNTY MEDICAL SOCIETY

Chairman: Dr. P. H. Kilbourne, Fidelity Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel. Time: 6:30 p. m., monthly, first Tuesday from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. L. de G. Joubert, 690 Dunlop St., Montreal, Canada.
Secretary: Dr. K. B. Johnston, 1509 Sherbrooke St., W., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Guy Maness, 119–7th Ave., Nashville, Tenn.
Secretary-Treasurer: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. James W. White, 15 Park Ave., New York.
Secretary: Dr. Rudolf Aebli, 30 E. 40th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

New York Society for Clinical Ophthalmology

President: Dr. Percy Fridenberg, 38 W. 59th St., New York.
Secretary: Dr. David Alperin, 889 Park Place, Brooklyn.
Place: Squibb Hall, 745–5th Ave. Time: 8 p. m., first Monday of each month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. Philip Romonek, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Ophthalmological Society of the University of Pittsburgh

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Time: Second Monday in November, January, March and May.

Passaic-Bergen Ophthalmological Club

President: Dr. R. N. Berke, 430 Union St., Hackensack, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Walter I. Lillie, 255 S. 17th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Edward Stieren, Union Trust Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. N. H. Turner, 200 E. Franklin St., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. Roy E. Mason, Frisco Bldg., St. Louis.
Secretary: Dr. Leslie Charles Drews, 508 N. Grand Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Russell Fletcher, 490 Post St., San Francisco.
Secretary: Dr. Avery Morley Hicks, 490 Post St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except May, June, July and December.

SHREVEPORT EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. R. Kirkpatrick, 6th and Walnut Sts., Texarkana, Ark.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. P. B. Greene, 422 Riverside Ave., Spokane, Wash.
Secretary: Dr. O. M. Rott, 421 Riverside Ave., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

SYRACUSE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. James F. Cahill, 428 S. Salina St., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 713 E. Genesee St., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. A. Lloyd Morgan, Medical Arts Bldg., Toronto, Canada.
Secretary: Dr. W. R. F. Luke, Medical Arts Bldg., Toronto, Canada.
Place: Academy of Medicine, 13 Queen's Pk. Time: First Monday of each month, November to April.

WASHINGTON, D. C., OPHTHALMOLOGICAL SOCIETY

President: Dr. G. Victor Simpson, 1710 Rhode Island Ave., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Frank D. Costenbader, 1726 I St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.